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**REVIEW OF
NEUROLOGY AND PSYCHIATRY**

REVIEW
OF
NEUROLOGY AND PSYCHIATRY

EDITOR

ALEXANDER BRUCE
M.D., F.R.C.P.E., F.R.S.E.

ASSISTANT EDITORS

EDWIN BRAMWELL
M.B., F.R.C.P.E. AND L., F.R.S.E.

CHAS. MACFIE CAMPBELL
M.B., CH.B.

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Contents

ORIGINAL ARTICLES.

	PAGE
PSEUDO-MYASTHENIA OF TOXIC ORIGIN. (PETROL FUMES.) By Sir William P. Gowers, M.D., F.R.S.	1
A NOTE ON THE CONDITION OF THE POSTCENTRAL CORTEX IN TABES DORSALIS. By Gordon Holmes, M.D.	5
CLINICAL AND ANATOMICAL DIAGNOSIS OF THE ANKYLOSING DISEASES OF THE SPINAL COLUMN. By Dr André Léri of Paris	12
CLINICAL AND ANATOMICAL DIAGNOSIS OF THE ANKYLOSING DISEASES OF THE SPINAL COLUMN—Part II. By Dr André Léri of Paris	65
THE EPICONUS SYMPTOM-COMPLEX IN CEREBRO-SPINAL SYPHILIS. By William G. Spiller, M.D.	77
ON THE SPINAL CHANGES IN A CASE OF MUSCULAR DYSTROPHY. By Gordon Holmes, M.D.	137
THE MYASTHENIC REACTION EXPERIMENTALLY PRODUCED IN THE FROG. By J. A. Gunn, M.D., B.Sc., M.A.	150
THE CYTOLOGICAL STUDY OF THE CEREBRO-SPINAL FLUID BY ALZHEIMER'S METHOD, AND ITS DIAGNOSTIC VALUE IN PSYCHIATRY. By Henry A. Cotton, M.D., and J. B. Ayer, Jr., M.D.	207
EXOPHTHALMIC GOITRE COMBINED WITH MYASTHENIA GRAVIS. By George E. Rennie, M.D., F.R.C.P. Lond.	229
A CASE OF ACUTE ASCENDING PARALYSIS OF SYPHILITIC ORIGIN. By O. Crouzon and Georges Villaret	275
A NOTE ON AN ASSOCIATED MOVEMENT OF THE EYES AND EARS IN MAN. By S. A. K. Wilson, M.B., B.Sc., M.R.C.P.	331
A CASE OF PARTIAL PTOSIS WITH EXAGGERATED INVOLUNTARY MOVEMENT OF THE AFFECTED EYELID: THE "JAW-WINKING" PHENOMENON. By William George Sym, M.D.	337
A CASE OF DISEASE OF THE POST-CENTRAL GYRUS ASSOCIATED WITH ASTEREOGNOSIS. By Purves Stewart, M.A., M.D., F.R.C.P.	379
A CASE OF SPASMODIC SYRINGOMYELIA (?). By Alexander Bruce, M.D., F.R.C.P.E.	390

	PAGE
ANEURISM OF THE ANTERIOR CEREBRAL ARTERY, WITH UNUSUAL PROLONGATION OF LIFE AFTER RUPTURE: AUTOPSY. By Alexander Bruce, M.D., F.R.C.P.E. ; J. H. Harvey Pirie, B.Sc., M.D. ; and W. Kelman Macdonald, M.B., Ch.B.	449
A CASE OF ARTERIO-VENOUS ANEURISM OF THE INTERNAL CAROTID ARTERY AND CAVERNOUS SINUS. By Alexander Bruce, M.D., etc.	462
CASES ILLUSTRATING THE COURSE AND PROGRESS IN DISSEMINATE SCLEROSIS. By W. B. Warrington, M.D., F.R.C.P. Lond.	521
THREE CASES OF HEMIPLEGIA FOLLOWING SCARLET FEVER. By J. D. Rolleston, M.A., M.D., Oxon.	530
A CASE OF INTRACRANIAL TUMOUR. By Edwin Bramwell, M.B., F.R.C.P.	577
LEPTOTHRIX INFECTIONS. A CASE OF PYÆMIA WITH MENINGITIS, AND NOTES OF TWO SIMILAR CASES. By E. Scott Carmichael, M.B., F.R.C.S.E.	631
THE EXAMINATION OF CEREBRO-SPINAL FLUID IN GENERAL PARALYSIS FOR PURPOSES OF DIAGNOSIS. By Hamilton C. Marr, M.D., F.F.P.S.G.	635
TUMOUR MALFORMATIONS OF THE CENTRAL NERVOUS SYSTEM. By William G. Spiller, M.D.	677
A RAPID METHOD FOR STAINING THE MYELINE IN NERVE FIBRES OF THE BRAIN AND SPINAL CORD (SIMPLE FORMOL OR FORMOL SULPHATE, FREEZING, ALUM-HÆMATEIN). By Dr J. Nageotte	682
ON THE ORIGIN OF THE FACIAL NERVE. By Alexander Bruce, M.D., F.R.C.P.E., and J. H. Harvey Pirie, M.D., B.Sc.	685

Review **of** **Neurology and Psychiatry**

Original Articles

PSEUDO-MYASTHENIA OF TOXIC ORIGIN. (PETROL-FUMES.)

BY SIR WILLIAM P. GOWERS, M.D., F.R.S.

THE following short account of a mysterious case is published in the hope of eliciting the experience of others regarding similar symptoms and the effects of the conjectured cause.

The patient was a major in the army, æt. 38, married for 12 years, who had never consciously suffered from venereal disease, and had run very few risks. There was no history of preceding grave illness or of any sore throat. Six months before he was seen he experienced, for a short time, a peculiar perversion of taste, by which all sweet things had a salt taste. It soon passed away, but since then he had experienced a tight feeling in the throat when swallowing. This became worse about six weeks before he was seen, and at the same time a definite difficulty in swallowing came on, and rapidly increased, until he became unable to swallow anything except such things as jellies or soups, which, as he expressed it, could "go down by themselves." Solid food he could not swallow, nor could he chew, although the masseters seemed to contract fairly. There was no nasal character of voice, and the palate was raised equally, though not vigorously; fluids did not regurgitate through the nose.

His speech presented a peculiar myasthenic feature, which had also come on during the preceding six weeks. When he

began to speak his voice was good and his articulation fair. As he went on speaking, his voice became feeble and his articulation imperfect. He felt, and seemed to have, great difficulty in moving his tongue and lips, and after three or four minutes his articulation became so imperfect that his speech was unintelligible. The first failure was in the softness of the guttural k, and in the linguals and dentals, such as l and r. He could whistle after rest, but not after a few minutes' speaking.

The eye-movements, internal and external, were normal, but the orbiculares palpebrarum were very weak, and opposed hardly any resistance to the separation of the closed lids. Moreover, there was little outward movement of the angles of the mouth when he smiled. No difference from the normal could be found in the electrical reactions. Sensation was everywhere normal, and prevented proper testing of fatiguability in the face by electricity. The optic discs were clear. He was easily tired in walking, but all reflex action was normal, and the knee-jerks were not lessened by long reproduction.

The pulse was 64 and the heart normal, the sounds only a little weak. He had had some peculiar attacks of pain in the chest, a sensation of a painful strain across the level of the lower part of the sternum, passing down to the neighbourhood of the umbilicus and up to the throat. No other symptom attended them, and they were not frequent. Otherwise everything was normal, and his habits had always been temperate.

The case clearly bore a very grave aspect. The resemblance of the symptoms to myasthenia was striking: the quickness of exhaustion of the muscles of speech and the feebleness of deglutition, the weakness of the orbicularis palpebrarum, and the peculiar smile. Although such symptoms have not been met with from a toxic cause, their onset in mid-life and rapid development suggested the possibility of this. Investigation failed to elicit any exposure to metallic poisoning, or sign of it, and inquiry into his occupation disclosed only one conceivable toxic influence. His work was in a Government factory, and he had for long been superintending the construction, and especially the testing, of petrol engines. He was constantly engaged in examining their working, which was done in a closed shed, and he had been continually exposed to the fumes of burned petrol, sometimes, perhaps, imperfectly burned. (See note below.)

He was then, of course, unable to work. He was ordered a hypodermic injection of strychnine nitrate, $\frac{1}{15}$ gr., twice a day. After a week a distinct improvement was observed, and this went on. I saw him again three weeks afterwards. He was able to swallow a little solid food, and even chew it. He could talk for five minutes without any impairment of articulation; then the tip of his tongue felt (to him) hard, and the linguals became imperfect, but he could speak, with rests, all day. The orbiculars were perceptibly stronger, and the angles of the mouth were better moved outwards in the smile.

His subsequent improvement was so steady, and his recovery apparently so complete, that his medical attendant thought it unnecessary for him to see me again. After some months he resumed his work.

But, just a year after I first saw him, he was brought to me again. The work he had returned to was nearly the same, and involved the same exposure to the petrol fumes. The old symptoms were then returning: the same ready fatiguability of the tongue and lips, and similar attacks of thoracic pain. He could speak well for four or five minutes, but then the tongue seemed to him to get tired, and he could not move it quickly in the mouth, and his linguals and dentals became faulty. If he did not stop, the enunciation became hardly intelligible. No difficulty in swallowing had developed.

He was ordered to relinquish permanently the work he had been doing, and resume strychnia in a pill. I have not seen him again, but subsequently learned that his symptoms soon passed off, and that he afterwards went to South Africa. It is two years since the relapse, and had he suffered again, it is practically certain that I should have seen him.

The case is remarkable from the close resemblance of the symptoms to those of partial myasthenia—partial in being chiefly conspicuous in the region of the bulbar nerves. The weakness of the orbicularis palpebrarum was not associated with any affection of the ocular nerves, but presented a striking correspondence with myasthenia, while the quick exhaustion of the power of articulation was even greater than is usual in that affection.

The course of the symptoms is thus more than suggestive of a toxic influence, connected with his occupation, as their cause,

but the only conceivable agent that could be heard of was the petrol fumes to which he had clearly been exposed. It was probable that the combustion of the petrol had sometimes been imperfect, and we do not know the nature, or deleterious character, of the products of its imperfect combustion. I could not, however, hear of any affection of other workers in the shed. This fact is of small importance, for it is common for one individual among many to have a special susceptibility to a given influence, and a special degree of exposure may have been involved in the close supervision he had to give.

Polyn neuritis affecting the legs has been observed from the habitual inhalation of carbon monoxide (*e.g.*, Glynn, *Brit. Med. Journ.*, i., 1895), but I have found only one case bearing directly on the effect of petrol fumes (Massanek, of Buda-Pesth; see *Neurol. Centralbl.*, 1904, p. 125). Two boys were shut up in a room in which a petroleum lamp was burning and smoking. One was dead, and his blood showed CO-hæmoglobin. The other, in a few days, developed an idiotic condition with signs of right hemiplegia. These soon lessened, but were followed by the symptoms of peripheral neuritis, weakness of the arms, and paralysis of the legs, with loss of the knee-jerks. This diminished after a few days, and had passed away at the end of six weeks. The case has importance as showing that petrol fumes may act on the nervous system, and makes this influence less improbable in the case I have described.

NOTE.—In a recent number of the Engineering Supplement of the *Times* (December 4, 1897) is an article on "The Exhaust Gases from Petrol Motors," by Prof. F. W. Burstall. He speaks of the great frequency in which the exhaust products from motor-cars have a very strong and disagreeable smell. He traces it to imperfect combustion of the vapourised petrol, which is difficult to avoid when the engine is run to obtain the maximum power, and to the fact that some of the petrol often comes over, as a fine spray, much of which is not perfectly burned. "When combustion commences the higher hydro-carbons do not necessarily at once burn to steam and carbon dioxide, but probably pass through a number of intermediate changes, forming the gases methane, acetylene, and a number of other hydro-carbons. This is more particularly the case if it should happen that any fluid is present in the form of very fine spherical globules, the outer skin of which burns, but the inner layer is unburnt. These gases, together with a considerable percentage of carbon monoxide, are thrown out from the motor, and give rise to the unpleasant smells."

These facts certainly afford confirmation of the suspicion that the products of imperfect combustion are various. They are

evidently of unknown nature, and can hardly fail to share, with carbon monoxide, noxious properties.

A NOTE ON THE CONDITION OF THE POSTCENTRAL CORTEX IN TABES DORSALIS.

By GORDON HOLMES, M.D.

IN his well-known monograph on "Histological Studies on the Localisation of Cerebral Function," Dr A. W. Campbell has described definite pathological changes in a portion of the cortex of the postcentral or ascending parietal gyrus in three cases of tabes dorsalis. This observation is of much wider interest and greater importance than the mere discovery of a new feature in the morbid anatomy of this disease, for, as tabes is, the author argues, essentially a sensory disease, the conclusion may be drawn that this area is "the primary terminus or arrival platform for nerve fibres conveying impulses having to do with 'common sensation.'"

If changes were found constantly in this area in uncomplicated cases of tabes, and if they occur only in this or in other affections and diseases of the sensory system, this would be a justifiable and logical conclusion. Its significance is increased by the fact that, while we have acquired the fairly extensive knowledge of the cortical motor centres which we possess by the experimental method, it does not at present seem possible by any means at our disposal to map out accurately the sensory centres by experiment on animals. This important task then falls to the lot of clinico-pathological investigation, and the difficulties and imperfections of this method are manifest.

The changes which Campbell has described bear on both the medullated fibres and the nerve cells of this area, but from his descriptions it appears that the alteration in the cells is the more prominent. This alone will be referred to in this note. These changes consisted in a noticeable reduction in the breadth of the cortex, a remarkable deficiency in cells of large size, a difficulty in defining the cell lamination, and a huddling together and a distorted arrangement of the cells. These changes

involved all layers of the cortex, but they were most prominent in the supra- and infra-stellate layers of large pyramidal cells, and especially in the former, in which there was a marked numerical deficiency of the larger cells, and apparently pathological distortion and shrinkage of those remaining. These changes were, however, not found throughout the whole area of the postcentral type of cortex, but were limited to the Rolandic or anterior wall of the postcentral gyrus and the corresponding area in the paracentral lobule. No changes were found in the cortex of any other region.

In reviewing Campbell's descriptions and his important conclusion there are a few points which must be considered. In the first place, if these changes in this limited area of cortex are, as the author assumes, secondary to the degeneration of the sensory protoneurons, the ascending morbid process must be interrupted in at least two masses of grey matter: in the dorsal horns of the cord or the dorsal column nuclei, and, finally, in the optic thalamus. No definite changes are, however, constantly found in the secondary sensory tracts or in the thalamus in *tabes dorsalis*. Further, it is *a priori* improbable that disease of even the final link of the sensory system—the thalamo-cortical fibres,—and it has never been demonstrated, should be able to produce such pronounced changes in the cells and intrinsic fibres of a mass of grey matter to which it is only afferent. As far as I am aware, there is very little evidence from experimental pathology that the cells of a centre suffer so severely, except where there has been a considerable lesion of its afferent fibres, or when the destruction has occurred in early life. The author attempts to escape this difficulty by assuming that the cortical changes which he has described may be an effect of "the complete interruption of the physiological impulses" which the degeneration of the dorsal spinal roots involves. But the sensory loss in *tabes* is rarely if ever complete in even a limited area of the body.

In the second place, if the area which Campbell has described as diseased in *tabes* is the centre of common sensation, the amount of change in this area and its extent should bear some relation to the degree and the distribution of the sensory loss in the case examined. Unhappily no mention is made of the state of sensation in the three cases which Campbell investigated.

And finally, if this cortical change depends on a structural alteration which ascends the sensory system, crossing over at least two synapses, and finally leads to degeneration or atrophy of the *cells* of the terminal receptive centre, it is necessary to postulate that the primary disease, that of the sensory proto-neurons, has been of considerable duration. Campbell's first two cases escape this criticism, as in the one the symptoms of tabes had lasted sixteen, in the other eight years; but in the third, which was a case of tabo-paralysis, the symptoms were of only fourteen months' duration, and yet the "typical" cortical changes were apparently as marked as in the others.

But this important question must be decided by the further examination of the cortex in tabes dorsalis and not by such theoretical considerations. I have had the opportunity of examining the brains of four cases of tabes. For the first two cases I am indebted to the kindness of Dr J. R. Lunn; the third case was on two occasions in the National Hospital under the care of Dr Ferrier, who kindly permits me to refer to the clinical notes which were then taken; the fourth case will not be referred to in detail, as it was only one of incipient tabes with very little sensory disturbance and relatively little degeneration of the dorsal spinal roots and of the dorsal columns of the cord.

Only two points in the clinical features of each case may be referred to, namely, the duration of the disease and the nature and the distribution of the sensory disturbance which was observed during life.

CASE I.—E. D., female, aged 45 years, had suffered with tabes for at least two years before death. The only noteworthy feature in the case was the presence of a large but painless arthropathy in one hip-joint. Through the kindness of Dr Lunn I was able to examine her some weeks before her death, which was due to pulmonary tuberculosis, but owing to her weak condition the examination could not be made complete. There was then marked analgesia of the inferior extremities, especially in the distal segments, a certain amount of anæsthesia, and practically complete loss of the sense of position and of muscle pain sense in these limbs. There was also practically complete analgesia and relative anæsthesia on the upper portion of the trunk, extending as high as the second rib and down the ulnar

borders of the arms, and definite loss of the sense of position in the distal segments of these limbs.

CASE II.—F. C., a female, was 43 years of age at death. In this case the disease had lasted about five years. Shooting pains were apparently a very pronounced symptom, but the only sensory loss referred to in the notes was anæsthesia and analgesia on the right hand and on the right side of the face. Attention was apparently directed to those regions by the coincident atrophy of the intrinsic muscles of that hand and the wasting of the right masseter and temporal muscles.

CASE III.—A. M., a male, was 49 years of age when he died in October 1906. He had had symptoms of tabes for at least sixteen years. When examined in January 1905, there was marked diminution of pain sensation everywhere below the level of the second rib and along the ulnar borders of the arms, complete tactile loss on the trunk between the nipples and the groins, absence of muscle pain sense and of the sense of position in the legs and diminution of both in the arms, and complete loss of bone sensation to a slowly vibrating tuning-fork in the lower limbs. He was again under observation in April 1906; the loss of all forms of sensation below the level of the second ribs and along the ulnar borders of the arms was still more marked.

In all these cases the ordinary routine was followed in the examination of the cortex of the central convolutions. The brain was hardened whole in 10 % formaline in normal saline solution, and after a few days the portions selected for examination were cut out and placed in alcohol, and, after dehydration, embedded in celloidin. Sections 15 μ thick were stained in thionin. In each of the cases several portions of the post-central gyri of both hemispheres were examined, and the structure of the cortex was compared with the corresponding regions in several brains which were either normal or in which no disease of the cortical cells was present.

It may be at once stated that no definite pathological changes could be found in the cortex of any of the four cases of tabes, either in the postcentral area or in any of the other regions which were examined; but it must be admitted that

as attention was centred on the ascending parietal gyrus, the examination of the rest of the brain was not complete.

In every case there was a marked difference between the structure of the cortex of the summit and that of the Rolandic wall of the postcentral gyrus, and this difference corresponded more or less closely to that described by Campbell in tabes; but the difference is equally marked and, as far as I can see, identical with that which is found in the normal brain. In the latter it is so striking that any one who has seen it must be surprised that so careful an observer as Campbell has failed to describe it. Brodmann has accurately described the difference between the cortex of these two contiguous areas in man and the lower apes; in fact, he has differentiated two distinct types of cyto-architecture in the postcentral gyrus which together correspond to the superficial extent of Campbell's postcentral area. Brodmann's *type 1* may be loosely described as that which extends over the convexity of the ascending parietal gyrus, and which in structure corresponds to Campbell's postcentral type, while his *type 3* covers the Rolandic wall of this gyrus and is visible on the surface only as a narrow strip which bounds the precentral cortex, or the area gigantopyramidalis, posteriorly in the paracentral lobule. Contrasted with *type 1* the structure of *type 3* is distinguished by the narrowness of the cortex, in fact "diese Rinde gehört zu den schmalsten Typen des ganzen Cerebrum"; the large pyramidal cells of the supra-stellate layer are smaller and more closely packed together, the layer of stellate cells is also narrower, and in the infra-stellate layer very few large cells are found. It will be at once seen that the difference there is, according to Brodmann, between the type of cortex which covers the Rolandic wall of the postcentral convolution and that of the convexity of this gyrus, corresponds fairly closely to the difference which Campbell has described between the cortex of these two regions in tabes dorsalis, and which he attributed to pathological changes in the Rolandic wall. Further, on comparison of Brodmann's figures with the illustrations in Plate IX. of Campbell's monograph, it may be seen that there is a fairly accurate correspondence between the superficial extent of Brodmann's *type 3* and the "affected tabetic area" of the English author.

In the brains of the four cases of tabes which I have

examined, as well as in a large number of control preparations, I have made careful comparison between the cortex of the two regions, and I have, as already mentioned, failed to find any evidence of pathological change in either area in the tabetic brains.

Finally, it must be pointed out that Campbell, in illustrating the changes which he regarded as pathological, has unhappily not drawn the figures which he reproduces for contrast from the same region of the normal brain. In Plate VII. the "affected tabetic area" is drawn from halfway down the Rolandic side of the postcentral gyrus, the normal from the Rolandic lip of the same convolution; while in Plate VIII. the figure which illustrates the normal has been drawn from the posterior wall of the gyrus.

Mention may be made of a few points in the histology of the Rolandic wall of the postcentral convolution, especially with reference to Campbell's descriptions. As Brodmann has pointed out, the diminished breadth of the cortex of this region is one of its most striking features. I have measured it and the cortex from the convexity of the same gyrus, in a large number of specimens, and have found that the relative breadth of the former, as compared with that of the latter, varies between the ratios of 1 to 1.25 and 1 to 1.5. [Fig. 3.] Of course for this purpose only sections were taken in which the cortex was cut absolutely vertically. There is a considerable variation in the depth of the cortex of each region: the figures just given have been arrived at by taking the average of a large number of observations. In Brodmann's photomicrographs (Bd. IV., Tafel 6, Figs. 1 and 3), which represent the cortex of these two regions under the same magnification, the breadth of *type* 3 in proportion to that of *type* 1 is approximately as 1 to 1.4.

In my cases of tabes the relative breadth of the cortex of the two areas did not exceed the normal limits.

My observations on the character and the arrangement of the cells in the area under consideration coincide so closely with those of Brodmann that detailed description is unnecessary. Attention may be, however, drawn to two points. In the first place, that the cortex of the Rolandic wall of the postcentral gyrus is not by any means a uniform layer: there may be considerable variations in its breadth over even a short stretch,

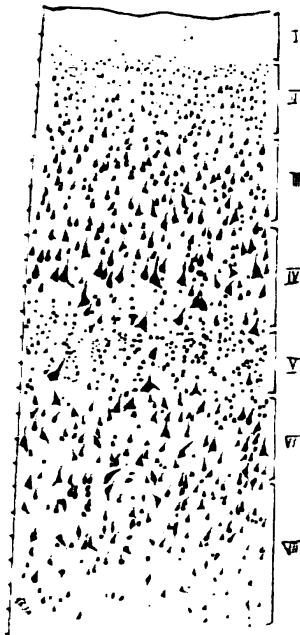


FIG. 1.

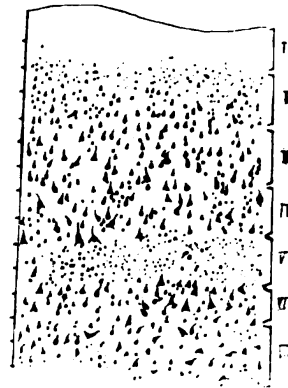


FIG. 2.

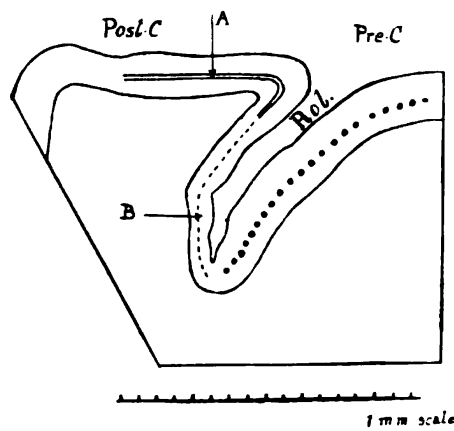


FIG. 3.

and the number of larger cells, especially in the supra-stellate layer, which are contained in it at different points, is far from constant. Campbell, too, has observed these irregularities in its cortical architecture. "The area could not be described," he says, "as a continuous and uninterrupted strip of diseased cortex, because, more or less healthy patches running through two or three sections were come across here and there." In addition to their small size, the cells of this region, and especially those of the supra-stellate layer, are often irregularly arranged, and not infrequently cells are seen in which the apical dendrite is widely deflected from the vertical direction.

From these facts the conclusion must be drawn that Campbell was in error in regarding the difference, which he observed in tabes between the structure of the anterior wall and of the summit of the postcentral gyrus, as due to pathological changes in the former, and that though the clinical and experimental evidence which indicates that the sensory centres lie behind the fissure of Rolando is ever growing stronger, we have as yet no proof that the centre for "common sensation" is co-extensive with the distribution of any type of cortex, or with any structural area.

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Campbell, A. W. "Histological Studies on the Localisation of Cerebral Function," Cambridge, 1905.

DESCRIPTION OF FIGURES.

- FIG. 1.—Drawing with the aid of a Leitz drawing-ocular from the convexity of the postcentral gyrus, just below the superior genu of the fissure of Rolando. It was taken from the point marked A in Fig. 3.
FIG. 2.—Drawing made with the same magnification as Fig. 1, from the Rolandic wall of the postcentral convolution in the position marked B, Fig. 3. It shows the reduction in breadth of the cortex, its less distinct lamination, and the absence of larger cells from layers iv. and vi.
FIG. 3.—A section of portions of the precentral and postcentral gyri, just below the level of the superior genu of the fissure of Rolando, drawn accurately to scale. It shows that the cortex on the Rolandic wall is narrower than that on the apex of the postcentral gyrus, the positions in the sections from which Figs. 1 and 2 were drawn, and the extent of the two types of cortex in the postcentral area. The double line represents Brodmann's type 1, the broken line his type 3.

CLINICAL AND ANATOMICAL DIAGNOSIS OF THE ANKYLOSING DISEASES OF THE SPINAL COLUMN.

By DR ANDRÉ LÉRI of Paris.

ONLY a few years ago all the affections of the spinal column that were accompanied with more or less pain, and with a greater or less tendency to ankylosis and which were not manifestly due to tuberculosis were ascribed to rheumatism. The word rheumatism was a convenient term under which were grouped promiscuously all articular or peri-articular diseases. It even included many conditions which affected the shafts of the long bones. Confusion was bound to attain its maximum in regard to diseases of the vertebral column, formed as it is of a long series of small bones and small articulations, the lesions in which cannot be clinically differentiated, and perfect anatomical demonstration of which is surrounded by numerous difficulties.

With regard to the limbs, the progress of clinical medicine and, up to a certain point, of bacteriology have clearly separated acute articular rheumatism, a disease apparently microbial in origin, from various other forms of acute rheumatism, and from all the varieties of "chronic rheumatism" which have a different pathogenesis; but the clinical subdivisions which have been made, and which are still incomplete, took into account for the most part the anatomical differences previously recognised. With regard to the spine, various clinical forms have been successively picked out from the chaos of "spinal rheumatism," and subsequently anatomical examination has justified the creation of these morbid entities. (It is mainly in the group of chronic and ankylosing affections that Professor Pierre Marie has been able to distinguish new and quite independent clinical varieties, by no means rare as a matter of fact, but previously misunderstood by the writers who had occasion to observe them because they had not been sufficiently isolated and described. We do not require further proof than the frequency of *spondylose rhizomélisque*, of which in the year following its description (1898)¹ we were able to collect more than thirty cases published previously under

¹ Pierre Marie, *Soc. Méd. des Hôp de Paris*, Feb. 11, 1898, et *Rev. de Méd.*, April 1898.

André Léri, "La Spondylose Rhizomélisque," *Rev. de Méd.*, Aug.-Sept., Oct., 1899.

various names, and of which a few years later there had been published hundreds of cases, examined in all countries; at present the observations have become so common that for the most part they are not published.)

Along with Professor Pierre Marie, we have devoted ourselves to the anatomical differentiations of these morbid forms, and the examination of the spinal columns has shown very manifest differences between each, and has at the same time thrown light upon their pathogenesis. It is especially to the clinical, and then to the anatomical descriptions of these various chronic ankylosing diseases of the spinal column that we shall devote this article.

I. CLINICAL DIAGNOSIS.

1. *Spinal Rheumatism*.—We may say that in the great majority of cases, the involvement of the spine in chronic deforming and ankylosing rheumatism occurs at a late stage of the disease. The small articulations of the extremities, especially of the hands and feet, are always, or almost always, the first to be affected. Chronic deforming rheumatism is essentially *acromelic*.

When later the spinal column is attacked in its turn, and more or less ankylosed, it is almost always by the examination of the deformities of the hands, which are so well known and so characteristic, that one can at a glance give the diagnosis of true "spinal rheumatism."

Pain in the spinal column may be essentially variable both in intensity and especially in site: it may from the very first extend through the whole of the spinal column; it may be and may remain localised to one of its divisions, and the cervical segment seems to be the region of predilection. The ankylosis which subsequently develops may also be generalised; more frequently it is localised either in the cervical region or in the lumbo-dorsal region. When it involves the whole spine the head of the patient may be bent forwards to a more or less marked degree, and the spine may present a more or less marked cyphosis; but in other cases the head may be erect and the spine may be absolutely upright, straight and rigid as a bar of iron, even with obliteration of the slight normal antero-posterior curves.

The rest of the trunk is not materially deformed when the

spine is straight; when the latter is curved, the thorax frequently presents a dilatation of variable extent, in the form of a barrel, more or less analogous to that found in emphysema; but there is never a marked antero-posterior flattening (Fig. 1).

The spine is frequently affected after the small joints of the extremities, without the large joints being markedly affected, and ankylosis of the spine does not in any way presuppose ankylosis of the hips or knees. In a general way one may say that the affection evolves by a succession of attacks, almost without any order, except that it commences in the small joints of the limbs. With regard to the attacks of pain, they are almost always very severe and frequently present the characters of intercostal neuralgia.

The subjects of the disease are most frequently already up in years, men or women, perhaps most often women. In their antecedents we may sometimes discover an infection which we may regard as causal. More frequently it can only be said that they belong to the class of "*grands neuro-arthritiques*."

2. *Spondylose Rhizomélisque*. — *Spondylose rhizomélisque*, first noted by Strümpell, described by Pierre Marie in 1898, and studied by Léri more particularly in its clinical evolution in 1899, presents a quite different clinical picture.

Its subjects are generally young, either adolescent or in the first part of adult life, up to the age of 35 or 40. In almost all cases it is men who are affected. They are not generally neuro-arthritics; but we find in the previous history of almost all of them evidence of the infection of which the existing group of ankylosing symptoms would appear to be the consequence. This infection is frequently tuberculosis, much more frequently still gonorrhœa; sometimes it is one of a variety of other infections. Moreover, very commonly close examination of the patient shows that the causal infection is still active; that the tuberculosis is frequently still in evolution, sometimes after a longer or shorter period of remission, which might have been mistaken for recovery. The gonorrhœa has usually left as a residue, either a morning gleet, or at least a slight turbidity of urine, indicating the presence of a very mild cystitis. It is, above all, after repeated and prolonged attacks of gonorrhœa that *spondylose* is to be observed.

The ankylosis is limited to the spinal column, to the hips and

shoulders, often to the knees ; frequently also to the sternoclavicular and temporo-maxillary joints. This does not imply that the pains, at the very commencement, may not be more or less generalised ; but the ankylosis never attacks the joints of the extremities, with the exception of some very rare cases, in which, *at a very late period*, it may also involve the small joints of the limbs ; but in general the ankylosis is clearly *spondylotic* and *rhizomelic*.

The evolution of the ankylosing process is variable, We may, however, draw up a general scheme which corresponds to the majority of the cases. In the first period of the disease the pains occupy the sacro-coccygeal region and are frequently very violent and persistent there. When they cease, we find that the lumbo-sacral column has become rigid. The hips become ankylosed almost at the same time, and with or without local pains. They may become ankylosed in more or less complete extension or in demi-flexion, perhaps according to whether the patient can or cannot continue to walk. It is this variable flexion of the thighs on the pelvis that gives to the patient the attitude which we have called the "flexion type," or the "extension type" (Figs. 2 and 3).

To this period of "inferior ankylosis," there usually succeeds a period of remission, during which the dorsal region becomes ankylosed in its turn, but in a more or less painless way, in any case without the violent intercostal pains which are frequently observed in spinal rheumatism.

Then comes the period of "superior ankylosis," characterised by violent pains in the cervical region, followed by ankylosis of this part of the spine. The neck then becomes bent to a degree which varies according to the case and according to the degree of flexion of the hips, but it never remains straight as we sometimes observe in spinal rheumatism.

If at this period we ask the patient to separate his arms from the body, we see that this movement of abduction and elevation is almost always very limited, that the arms frequently do not reach the horizontal position. If a hand is placed on the shoulders, we discover coarse crepitations in the joint. The ankylosis is almost never complete at the shoulder joints, while on the other hand it frequently is so at the hip joints.

The knees are, as a rule, attacked later, and often to a slight

degree, so that sometimes one may only note that in extreme flexion the heel cannot come into contact with the buttocks. The sterno-clavicular and temporo-maxillary joints are frequently affected more or less early and more or less severely, generally with a certain degree of local pain.

The thorax is almost always flattened from in front backwards, the sternum being approximated to the spinal column and the ribs being more oblique than in the normal condition. This deformity is often very marked. The pelvis is also involved in the antero-posterior flattening, but this fact is more difficult to verify; it arises perhaps in part, but only in part, from a kind of slight tilt of the bones of the pelvic girdle. Radiography shows, however, a marked deformity of the pelvis, which, as a result of the subsidence of the spinal column, assumes the form more or less of a heart on playing cards.

The evolution of this affection is essentially chronic. Following the two principal stages of development (viz., that of pain and that of ankylosis), affecting first the lumbo-sacro-coccygeal, then the cervical region, there may occur further periods of pain, more or less acute, generally much less marked than the former, and mainly cervical in their localisation. But as a general rule the disease tends towards a stage of final ankylosis without pain; patients are left in this state of disablement without a tendency towards further progress of the disease.

3. *Hereditary Traumatic Cyphosis*.—Hereditary traumatic cyphosis, described by Bechterew as "ankylosing rigidity of the spinal column," and studied specially by Pierre Marie and Astié, presents an absolutely different clinical picture. Most frequently the subjects are already old, and in their hereditary antecedents, direct or collateral, we discover the tendency to abnormal spinal curvatures, cyphosis or hunchback. In most cases the disorder arose from a sudden injury in patients who already had a more or less marked degree of senile or pre-senile cyphosis. The accidents hitherto described have been of two kinds; either the patient has fallen on his back, perhaps while carrying a heavy load, or a weight has fallen on his back. We can understand that in either case the accident would tend to straighten the physiological dorsal curve, and still more so if the spinal column were already abnormally cyphotic. However that may be, during the days following the injury the dorsal pain becomes

somewhat violent, the cyphosis is very marked, and the patient stoops in walking. Then the pain abates, the patient straightens himself, and during the next few months walks sometimes more erect than before. Then slowly, gradually, with or without fresh pains, but without apparent reason, there appears a very marked dorsal convexity (*gibbsite*), not angular but semi-circular, and frequently of very small radius. This convexity occupies the extent of four, five, or six vertebræ; it cannot be reduced by suspension of the patient, whilst the cyphosis, which persists above and below it, can still be reduced. Thus it is clearly only at the level of the convexity that the vertebrae are ankylosed.

This ankylosis is not as a general rule accompanied by very severe pain, at least not by spontaneous pain. The vertebrae, however, remain for a long time painful to pressure at the level of the convexity. The thorax is always very much dilated from in front towards the back, as in all cases of hunchback (Fig. 4).

It would seem that the ankylosis is not always permanent, and a manifest improvement, if not recovery, may supervene after rest in bed and the wearing of a corset.

All the other joints are intact, and in short, this hereditary traumatic cyphosis behaves just like a local accident, and not, as in the preceding affections, like a general disease. It can be easily recognised with a little care. The etiological difficulty is constituted by the long period which has elapsed since the occurrence of the injury, but it is precisely its peculiar evolution in two parts which is one of the best diagnosing signs. Moreover, it is much more rare than spinal rheumatism and *spondylose rhizomélisque*.

A fact which it is important to note, however, is that in very rare cases of traumatic cyphosis there may appear signs of compression of the spinal cord which are more or less serious and persistent. Henle, in a case of this kind, observed a tendency to spasticity. We ourselves have had the opportunity of observing, in a case as yet unpublished, a true spastic paraplegia. Pathological anatomy has seemed to us to give the key to the explanation of these unusual and serious symptoms. We shall return to this question further on.

4. There exist without doubt many other varieties of non-tubercular vertebral ankyloses, but they are almost always more distinctly limited than the preceding forms, and the spinal ankylosis

is merely an accidental and limited localisation of a more general disease. As they are little known clinically and anatomically, it is unnecessary to do more than mention them.

Gout of the spine, described by Lécorché, is as a rule merely a localisation of the general affection, preceded for a considerable time by acute and repeated attacks in the small joints, especially that of the great toe, and frequently by various non-articular manifestations of gout. It presents two predominating localisations, the lumbar region and the cervical region. The uratic deposits cause severe local pains, crackings of the joints, symptoms of nerve compression, neuralgias, amyotrophies, paraplegias, but they do not lead to true ankylosis.

Syphilis of the spine, which appears to be very rare, produces a true "syphilitic Pott's disease," localised principally in the cervical region. Apart from the ulcerous form, analogous to tubercular Pott's disease, Leyden notes the existence of an osteophytic form, which would be very much analogous to chronic rheumatism, but as yet we have not sufficient pathological data to permit of our describing a spinal syphilitic ankylosing disease of the spinal column.

Various abnormal attitudes of the spinal column, resulting from conditions rather *physiological* than truly pathological, may give rise to confusion with the ankylosing diseases of the spine which we have described. Amongst these the *duplicature champêtre* (peasants' stoop), described by Pierre Marie, consists of a cyphosis with ankylosis of the lumbar region in the position of flexion. It is observed in field-workers who are constantly stooping over their spades or their ploughs. It is accompanied neither by ankylosis of the whole of the spine nor by *pronounced* ankylosis in the joints of the limbs (Fig. 5).

Senile cyphosis, a gradual curvature of the trunk which occurs in a great number of old people, and which may be easily recognised, has not, to our knowledge, been hitherto described, and has not been anatomically studied. Without as yet being very positive, we believe, from what we have been able to observe, that it results mainly from a real subsidence of the rarefied vertebral bodies (rarefied as, for example, is the neck of the femur in old people). It is this subsidence which in old people produces the shortening of the figure; which also, especially at the anterior part of the vertebral bodies (because it is there that, on



FIG. 1.



FIG. 2.



FIG. 3.



FIG. 4.



FIG. 5.

account of the normal dorsal curvature, they undergo the greatest pressure), produces a senile cyphosis which is sometimes very marked. The bodies of the vertebrae thus form an angle with each other, and we doubt whether there are produced in this case any true spinal ankyloses. It is for this reason, that apropos of the clinical study, we have made this incursion into the pathological anatomy. We reserve for the anatomical study which follows the three great ankylosing diseases of the spinal column, of which we have described the differential clinical characters.

(To be continued.)

DESCRIPTION OF FIGURES.

- FIG. 1.—Chronic Ankylosing Vertebral Rheumatism, with commencing ankylosis of the hip-joints. The deformities of the hands and the slight relative curvature of the vertebral column are to be noted.
- FIG. 2.—Spondylose Rhizomélisque, “flexion type.” Note the flexed position of the hip-joints.
- FIG. 3.—Spondylose Rhizomélisque, “extension type,” with in addition dislocation of the cervical column. Note the slight curvature of the spinal column, the flattening of the thorax, and the normal state of the extremities.
- FIG. 4.—Hereditary Traumatic Cyphosis; the extreme degree of curvature limited to the dorsal region.
- FIG. 5.—*Duplicature champêtre*, or Peasant's Stoop of Marie. Note the cyphosis in the lumbar region and the stooping attitude.
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Abstracts

ANATOMY.

ON CERTAIN PERIVASCULAR CORPUSCLES IN THE CEREBRAL SUBSTANCE.

- (1) **BREAL SUBSTANCE.** (Sopra speciali corpuscoli perivasali nella sostanza cerebrale.) U. CERLETTI (of Rome), *Riv. Sper. di Fren.*, Vol. xxxiii., Fasc. 2-3.

THE elements described by the author are found in the perivascular spaces in the cortex, both in normal and in pathological conditions, in animals as well as in man. They stain faintly, show no nucleus, have semilunar or ovoid shape, appear homogeneous or granular; they are most easily demonstrated by means of a pyronin, methylene-green stain (Unna-Pappenheim); they are morphologically distinct from the well-known ectodermal and mesodermal elements of the cortex; their meaning is not known. Nine figures are given to illustrate these perivascular bodies.

C. MACFIE CAMPBELL.

PSYCHOLOGY.

RESULTS OF FATIGUE MEASUREMENTS IN 64 SCHOOL-CHILDREN.

- (2) **CHILDREN.** (Ergebnisse von Ermüdungsmessungen an 64 Schulkindern.) EDUARD QUIRSFELD, *Prag. med. Woch.*, Oct. 24, 1907.

STIMULATED by the interest awakened by the results of the measurement of fatigue in school children made public by Dr Schuyten at the thirteenth International Hygienic Congress in Brussels (1903), Dr Quirsfeld undertook an investigation on similar lines.

The children selected were the brightest in the class, and were divided by the teacher into visual and auditive types. To secure their confidence, a preliminary set of memory tests was undertaken. The material used consisted of series of letters or numbers, mental division, etc. The following are the chief results obtained:—

When five seconds were allowed for writing down the remembered letters, the auditive children surpassed the visual; but when ten seconds were allowed, the visual children were superior. When numbers formed the material, the visual were always superior. The visual boy was better than the auditive in mental arithmetic, and the boys better than the girls.

For his experiments Dr Schuyten had used rows of figures, and by testing the memory for these rows had sought to compare the mental activity of children in the morning with that in the afternoon. The afternoon test was made one day and the morning one the following day. In both boys and girls the morning results were superior. Dr Quirsfeld repeated these experiments, taking the morning tests on the first day, the afternoon ones on the second; he found that in both boys and girls the afternoon results were superior. He then assured himself that, whether the exercises were begun morning or afternoon, the second result was invariably the better.

With twelve girls and twelve boys he then tried a series of similar experiments, taking the children on four successive days. The following figures, representing the intellectual activity of the scholars at the times mentioned, show the results obtained:—

	Boys.	Girls.
At the beginning of the forenoon instruction . . .	89·93 %	42·59 %
At the end " " " . . .	51·16 %	44·77 %
At the beginning " afternoon " " . . .	45·88 %	46·66 %
At the end " " " . . .	44·34 %	35 %

These results show that some hours' work improves the memory, but that fatigue does actually set in during the afternoon instruction, which must, therefore, be reckoned as of less value than that of the morning.

Dr Quirsfeld undertook another series of investigations, using a turning cylinder with numbers printed on it, and causing the children to add the numbers in pairs and say the sums aloud. Each trial lasted for fifteen minutes. Mistakes occurred most frequently in the second half of the period; the maximum number of additions was made between the sixth and tenth minutes, the minimum between the tenth and fifteenth. Visual children gave the best results, and the boys far surpassed the girls.

In conclusion, Dr Quirsfeld emphasises the desirability of a knowledge of psychology on the part of the teacher, and the necessity of instruction being suited to the enormously varying individualities of the children concerned.

MARGARET DRUMMOND.

THE SLEEP OF ANOTHER. (Der Schlaf des Andern.) Dr PAUL
(3) KRONTHAL. Carl Marhold, Berlin, 1907. Pp. 45.

THIS pamphlet is an expansion of a paper on Sleep delivered on November 5th 1906, to the Berlin Society for Psychiatry and Nervous Diseases.

The author begins by making clear his point of view. He thinks that the reason why scientific men have not yet agreed on

any theory of sleep is that they have treated it now from the subjective, now from the objective side, without clearly realising that in shifting their ground in this way they alter all the pre-suppositions on which their arguments are based. As soon as the subjective is introduced we pass to a world in which agreement is unattainable, because every man's sensations are known to himself alone, and by no possibility can be made known to another for purposes of investigation and comparison. Hence the man of science must confine himself exclusively to the objective; matter, energy, time, space, number, the laws of logic, and the law of causality form his postulates; and he must not inquire into the grounds on which these conceptions rest. When he has resolved a problem as far as is possible for the senses, then his work is done.

In accordance with this view Dr Kronthal proceeds to an examination of sleep as a purely objective phenomenon—"the sleep of another." His definition of sleep from this standpoint is, "the temporary condition of a living organism in which most of the reflexes are set aside or arrested." According to this definition the cessation of reaction which is brought about when a detached frog's muscle is stimulated continuously for some time is sleep. Moreover, the pathological states brought about by the action of chloroform, ether, etc., and cold arise not only in animals with highly developed nervous systems, but in organisms without nervous systems at all and in bits of muscles. Hence sleep is not dependent, as is commonly supposed, on the nerve cells of the brain; on the contrary, every cell in the body suffers from fatigue and periodically requires rest or sleep.

The importance of the nerve cell has, according to our author, been greatly exaggerated; every disease of which we know nothing has been set down as "nervous," in spite of the fact that in many such diseases, *e.g.*, hysteria, mania, melancholia, epilepsy, the nervous system may be perfectly healthy. As a matter of fact the central nervous system has no special directing authority, but is merely an office by means of which stimuli are transmitted to the motor apparatus.

If the definition given is a true one, then sleep should occur (1) when stimuli fail, (2) when the sense organs do not act, (3) when the conduction of stimuli to the motor apparatus ceases. In all these three cases we find that sleep actually does arise, thus confirming the truth of the definition; but in only one of them, the third, is the condition brought about by the state of the nervous system. The brain being the central office, whither sensory paths converge, is the only place where an injury can interfere sufficiently with conduction to arrest most of the reflexes and so give rise to sleep.

The attribution of special directive agency to the part of the nervous system containing the most nerve cells, viz., the brain, Dr Kronthal attributes to the belief that the soul is situated in the brain and thence directs the body. He proceeds therefore to inquire what, from a naturalistic point of view, is the meaning of the term "soul." Its meaning he finds to be simply "the sum of the reflexes": the more reflexes, the more soul. An infant, in whom comparatively few sensori-motor arcs are developed, has little soul. As we descend in the animal scale, the sum of the reflexes decreases, and corresponding to this we find less and less soul. The introduction and discussion of the term soul evidently arises from the felt necessity of unifying the reflexes. But in using the term soul in this way Dr Kronthal robs it of all the most important part of its content. A writer in the *Hibbert Journal* recently defined the soul as "that which feels," but feeling Dr Kronthal excludes altogether from his material. He expressly says, "Of feeling we can never know anything. Whosoever, therefore, conceives of will and feeling as faculties of the soul removes the soul to the realm of mysticism." We sympathise heartily with the writer in his endeavour to view his subject from one clearly defined standpoint; but we think it would be better if in so doing he rejected the term soul, with its ineffaceable connotation, as decisively as he rejects the term feeling.

In his attribution of fatigue to all the cells Dr Kronthal disagrees with Professor Mosso and other authorities, who regard it as being always nervous in character. The valuable and suggestive examination set forth in this paper might well be supplemented by an inquiry into what actually takes place in the individual cells when fatigue sets in.

The clear recognition of the postulates of science and of the special standpoint of the scientific man lends considerable philosophic interest to this treatise. We are, however, inclined to think that the author exaggerates the difficulty of comparing subjective experiences. After all, it is from our subjective sensations that we construct the whole spatial world in which measurement rules supreme, and the very fact that in this particular instance we all do construct the same world, gives good ground for the presumption that our more intimate worlds, where hope, fear, desire, and purpose hold sway, have also so much in common as to render understanding and comparison possible. The valuable results in psychology which have been obtained by the researches of Galton, James, and other students, are a standing proof that this presumption is justified.

MARGARET DRUMMOND.

PHYSIOLOGY.**ON AN APPARENT MUSCULAR INHIBITION PRODUCED BY****(4) EXCITATION OF THE NINTH SPINAL NERVE OF THE FROG, WITH A NOTE ON THE WEDENSKY INHIBITION.**

V. J. WOOLLEY, *Journ. of Physiol.*, Vol. xxxvi., Nos. 2 and 3, p. 177.

NICOLAIDES and Dontas showed that if a continuous tetanus of the frog's gastrocnemius is produced by a strong excitation of the eighth spinal nerve, then a weaker stimulation of the ninth will often bring about a relaxation. This was supposed by them to prove that the ninth spinal nerve contains inhibitory fibres to the gastrocnemius. The author believes, however, that this phenomenon is a particular case of the Wedensky inhibition, and that it is not due to the presence of inhibitory fibres in the ninth nerve.

SUTHERLAND SIMPSON.

STRYCHNINE AND REFLEX INHIBITION OF SKELETAL**(5) MUSCLE. C. S. SHERRINGTON, *Journ. of Physiol.*, Vol. xxxvi., Nos. 2 and 3, p. 185.**

THE "flexion-reflex" of the hind limb in the cat was used in this research. The femur was firmly clamped, the vasto-crureus (extensor) and semi-tendinosus (flexor) isolated, and one or other attached to a recording lever, the flexion-reflex being induced by stimulating faradically the afferent nerve—either a branch of the internal saphenous or the musculo-cutaneous division of the peroneal.

When this reflex is brought about, the flexor group of muscles (for the knee-joint) contract, and at the same moment there is a relaxation of the extensor group, this relaxation of the vasto-crureus being indicated by a fall of the lever. This relaxation is due to a central inhibition which quells any centrifugal discharge which may be exciting or maintaining contraction in the muscle. But following on this relaxation-phase of the extensor there is, if the exciting stimulus be strong or prolonged, a second phase of contraction indicated by an upward movement of the lever. The first, or relaxation-phase, lasts as long as the external stimulus is applied; the second, or contraction-phase, ensues on cessation of the external stimulus.

After strychnine, even in small doses, the relaxation-phase is abolished in the extensor part of the flexion-reflex and replaced by a contraction-phase, the normal second or rebound contraction-

phase being suppressed. The extensor part of the "flexion-reflex" comes therefore under strychnine to resemble the flexor part, both groups of muscles contracting at the same moment, and so the normal co-ordination is completely destroyed. Under chloroform and ether the strychnine effect disappears, and the normal simultaneous contraction of flexors and relaxation of extensors can be again brought about, but if the narcosis is remitted, the change induced by strychnine returns. SUTHERLAND SIMPSON.

**ON THE RELATION BETWEEN THE PHYSICAL, CHEMICAL
(6) AND ELECTRICAL PROPERTIES OF THE NERVES.**

Part I. N. H. ALCOCK and G. R. LYNCH, *Journ. of Physiol.*,
Vol. xxxvi., Nos. 2 and 3, p. 93.

In this preliminary communication the authors have estimated the percentage of water and of chlorine in the nerves of different animals (cat, dog, goat, horse), and in different nerves of the same animal, and they have also tried to determine the percentage solutions of NaCl and of KCl isotonic with these nerves, by immersing them in the above solutions and observing at what concentration they neither gain nor lose in weight. They summarise as follows:—

(1) The average percentage of water in the medullated nerves of cats is 67·3; dogs, 69·7; goats, 75·4; horses, 69·3; and for non-medullated nerve from the horse, 81·2.

(2) The percentage of water varies in different animals of the same species, and in different nerves of the same animal, *e.g.*, the internal popliteal nerve of the cat has invariably a higher percentage than the external popliteal.

(3) Nerves of the cat placed in solution of pure NaCl in distilled water of approximately 1·16 per cent. remain unchanged in weight. This differs from the 0·9 per cent. usually taken as isotonic with mammalian tissues.

(4) The average percentage of chlorine (0·23 per cent.) is the same for both non-medullated and medullated nerves of the horse. This does not agree with the results of Macallum and Menten, who state that the medullary sheath is practically free from chlorides. SUTHERLAND SIMPSON.

PATHOLOGY.

TRAUMATIC LESION OF THE PONS AND TEGMENTUM WITH
 (7) **DIRECT AND RETROGRADE DEGENERATION OF THE**
MEDIAN FILLET AND PYRAMID, AND OF THE HOMO-
LATERAL OLIVE. A. MEYER, *Journ. of Nerv. and Mental Dis.*,
 Nov. 1907.

A SHORT paper is given describing the above lesion as a consequence of an injury to the neck, causing apparently a fracture of the base of the skull.

JOHN D. COMRIE.

ON THE PATHOLOGY OF THE LENTICULAR NUCLEUS. (Sulla
 (8) **patologia del nucleo lenticolare.**) G. FRANCESCO (of Rome),
Riv. Sper. di Fren., Vol. xxxiii., Fasc. 2-3.

THE author reports the history of a patient with a hæmorrhagic cyst of the putamen, and describes the pathological anatomy with the secondary degenerations, illustrated by eight figures. He also refers to a case with a lesion of the retro-lenticular region. The following are his main conclusions:—

Hæmorrhage of the putamen, when the retro-lenticular region of the capsule is implicated, gives rise to a very complicated symptomatology, which often simulates that of a focal cortical lesion with a mental syndrome. The median fillet passes mainly into the lenticular nucleus, but is also connected with the thalamus through the hypothalamic nucleus; the lenticular nucleus is connected with the nuclei of Goll and Burdach of the opposite side through the hypothalamic nucleus and the median fillet. The pes lemniscus profundus is probably a trophic path. The commissure of Meynert is in relation with the lenticular nucleus, and has no connection with the optic tract. Pseudomelia parestesica is related to lesions of the lenticular nucleus or of the retro-lenticular region of the capsule. The putamen is an important trophic station in connection with long trophic paths which cross over in the cord. Lesions of the putamen can produce acute cerebral hemiatrophy.

C. MACFIE CAMPBELL.

SYRINGOMYELIC LESION IN A CASE OF CATATONIC
 (9) **STUPOR.** (Lésions syringomyéliques chez une catatonique.)
 G. DENY et A. BARBÉ, *L'Encéphale*, Sept. 1907, p. 283.

THE patient, a woman of 50, had been catatonic for seven years; no syringomyelic symptoms were noted during life. At autopsy the

cord appeared normal till cut. Sections revealed the presence of a cavity continuous with the central canal extending from about C 4 to D 10. The shape varied from an antero-posterior slit in the cervical enlargement, to an irregular polygon in the upper dorsal region; the posterior grey commissure was the only part of the cord which suffered much. The tissue around the cavity was of the usual gliomatous type. A peculiarity of the case was the presence in the cavity of small glial columns (like the columnæ carneæ), each containing a blood-vessel running their whole length. They argue in favour of the cavity being associated with a pre-existing anomaly of the central canal from the presence of an asymmetry of the grey matter in the cervical matter which they take to be a congenital malformation. From the illustrations one could not tell that it was more than collapse of the cord when the fluid escaped from the syringomyelic cavity.

J. H. HARVEY PIRIE.

A CONTRIBUTION TO THE STUDY OF "NEUROTOXIC (10) SERUMS" AND THE LESIONS OF THE NERVOUS SYSTEM INDUCED BY THEM—ISO-NEUROTOXIC SERUM.

(Contributo allo studio dei "sieri neurotossici" e delle lesioni da essi provocate nel sistema nervoso—siero iso-neurotossico.)

ROSSI, *Rev. di Patolog. Nerv. e Ment.*, F. 9, 1907, p. 417.

THE serum is prepared in the following manner. An animal is killed by bleeding from the carotid, the brain cut into small pieces, what little blood remaining being washed out by physiological solution. A quantity is then weighed, triturated into a homogeneous mass, and sterile physiological solution added until a determined quantity of nerve substance is obtained for injections. The emulsion so obtained is then filtered. All details are carried out with the strictest aseptic precautions. The emulsion is now injected into the peritoneum of an animal from which the toxic serum is to be taken. The injections are performed at intervals of 4 or 5 days, and repeated a varying number of times.

The animal is then bled 6 to 7 days after the last injection, the blood collected in sterile tubes and the serum then separated. Injection of this serum is performed directly into the nervous centres.

Conclusions.—1. The injection into an animal of a certain species, of nerve substance from another species, gives to the serum of the former a toxic power towards the nervous system of the latter. 2. The injection of grey or white substance, in the process of preparing the serum, does not give the same clinical results afterwards. 3. If one injects an emulsion of the brain of

a guineapig into a guineapig, the serum, after a certain number of injections, becomes neurotoxic to this animal. 4. The action of these sera is not quite specific. 5. All the properties of these sera do not correspond to those possessed by a typical cytolytic serum. 6. The anatomical changes induced are constant; the gravest are those of the nervous system. DAVID ORR.

PLASTICITY AND AMCEBROIDISM OF THE CELLS OF THE

(11) **SENSORY GANGLIA.** (*Plasticité et Amiboïsme des Cellules des Ganglions sensitifs.*) G. MARINESCO, *Rev. Neurol.*, Nov. 21, 1907, p. 1109.

As in the author's previous papers, the plexiform ganglion was transplanted under the skin of the ear (rabbit, auto-transplantation), and the modifications the cells undergo are again given in detail. But in this article Marinesco theorises as to the manner in which the surviving cells become morphologically altered, and ascribes the increase in volume of, and formation of expansions in the cells at the periphery of the ganglion, to movement. A certain number of these cells, besides throwing out protuberances and ramifications, appear swollen, indicating the penetration of liquids into their interior.

The production of these expansions is not due to variation of osmotic pressure, nor to accumulation of the products of disintegration and CO₂, which always raise the osmotic pressure, for such new formations are only found in the cells at the periphery of the ganglion, *i.e.* in those which survive the process of transplantation. DAVID ORR.

PERIVASCULAR INFILTRATION WITH PLASMA CELLS IN

(12) **THE CENTRAL NERVOUS SYSTEM OF ALCOHOLISED RABBITS.** (*Perivaskuläre Plasmazelleninfiltration, etc.*) J. MONTESANO (of Rome), *Centralbl. f. Nervenheil. u. Psych.*, Nov. 15, 1907.

THE presence of a diffuse perivascular plasma-cell infiltrate in the cortex is a very important differential point in the histopathological diagnosis of general paralysis. In chronic alcoholism the vessel walls show chiefly regressive changes, and the perivascular lymph-spaces show no infiltrate.

The author gave rabbits up to 12 ccm. absolute alcohol diluted with water daily, sometimes in conjunction with an intravenous injection of adrenalin, for variable periods; in four cases he found plasma-cell infiltration of the cortical vessels.

C. MACFIE CAMPBELL.

THE PATHOLOGICAL CHANGES IN THE CENTRAL NERVOUS**(13) SYSTEM IN EXPERIMENTAL DIPHTHERIA. CHARLES**

BOLTON and S. H. BROWN, *Brain*, Oct. 1907, p. 365.

THE authors confirm by their work previous results obtained by Crocq, Nouravieff, and Rainy on rabbits, and have also found that the nerve cells of the monkey are susceptible to the action of diphtheria toxin, and that the medullary centres of this animal are more definitely affected than are those of the rabbit, where the cells of the spinal cord are chiefly affected. The cerebrum, cerebellum, and spinal ganglia were in all cases normal, the nerve-cell degeneration being limited to the cord, medulla, pons, and mid-brain. The degenerate cells are scattered, not in any definite focus, and are mingled with others apparently normal. The changes in the cells are, on the whole, slight, although in some the degeneration is extreme.

They believe that the degeneration is a direct effect of the diphtheritic toxin, not secondary to circulatory changes; that the peripheral nerves are also directly affected, their degeneration not being merely a result of their central cells being affected; that the fatty degeneration of the heart is also direct, not merely subsequent to the medullary affection.

J. H. HARVEY PIRIE.

CLINICAL NEUROLOGY.**HEREDITY IN DISEASES OF THE NERVOUS SYSTEM, WITH****(14) SPECIAL REFERENCE TO HEREDITY IN EPILEPSY.**

PHILIP COOMBS KNAPP (Boston), *Boston Med. and Surg. Journ.*, October 10, 1907, Vol. clvii., p. 485.

OUR ideas as to heredity have been considerably modified by the doctrines advanced by Galton, Weismann, Mendel, and others. Weismann's theories indicate that morbid heredity occurs only when the germ plasma is pathologically altered by certain injurious causes, such as intoxication, infection, constitutional disease, or local disease of the generative tract, causing constitutional weakness of the individual. Galton and Mendel have emphasised the fact that when such morbid heredity does occur it tends to disappear in succeeding generations by the influence of new blood. The present data as to morbid heredity as a cause of nervous disease are of little value. Similar heredity is rare, and is seen chiefly in certain very uncommon diseases, such as Huntington's chorea, Friedreich's ataxia, muscular dystrophy, amaurotic idiocy, etc., although it is also rather frequent in migraine. Although a hereditary tendency is common in these diseases, many cases occur

without any such taint. Dissimilar heredity, in the form of the neuropathic or psychopathic predisposition, is thought to be much more frequent. The statistics of such heredity in insanity are invalidated by the fact that wholly different diseases are classed under the one heading of insanity and conclusions are then drawn. Epilepsy is a syndrome rather than a disease, so that the statistics as to epilepsy are open to the same objection, although in a somewhat less degree. The statistics as to neuropathic heredity vary enormously, from 11 to 87 per cent. In 1200 cases at the Massachusetts Hospital for Epileptics, neuropathic heredity was found in 14.5 per cent.; in 320 personal cases it was found in 35 per cent. Similar heredity was likewise found to vary from 0.9 to 37.2 per cent. It is possible that similar heredity has some influence. The existence of other forms of nervous and mental disease in the antecedents in a certain percentage of cases has been unwarrantably assumed to be evidence that morbid heredity was an important factor in the ætiology of nervous and mental disease. The special ætiology of these antecedent cases of nervous disease, however, must be considered, and cases due to trauma, infection, or toxic influences excluded. It is also unfair to assume that the existence of nervous disease in the antecedents is evidence of a morbid heredity, unless the percentage of nervous disease is greater than that occurring in the antecedents of healthy persons. Very few studies have been made to show how often nervous disease does occur in the antecedents of healthy persons, but Thomann-Koller and Diem have found it in 59 and 66.9 per cent. respectively, a percentage greater than has been reported in many of the collected cases of epilepsy. In many cases the morbid susceptibility of the patient is due not to a special nervous heredity, but to the fact that he is a weakling, the weakness manifesting itself in all parts of the body, the offspring of parents in whom the germ plasma was pathologically altered from some of the causes above indicated. The individual case must be studied on its merits, and the existence of nervous or mental disease in the antecedents is not to be accepted as proof of the hereditary character of the patient's ailment without thorough investigation of the ætiology and nature of such diseases in the antecedents. AUTHOR'S ABSTRACT.

ACUTE ANTERIOR POLIOMYELITIS, OR ACUTE SPINAL
 (15) **PARALYSIS OF CHILDREN.** (Remarks on the Epidemic
 now prevailing in New York.) JOSEPH COLLINS (New York),
Med. Rec., Nov. 2, 1907, p. 725.

EPIDEMICS of acute anterior poliomyelitis have been recorded by
 Medin (Stockholm, 1887), Looft (Norway, 1900), Leegard (Norway,

1901), Mackenzie (New York, 1902), Wade (Sydney, 1904), and Litchfield (Sydney, 1904).

Collins draws attention to what he regards as an epidemic at present in progress in New York. He states that at the out-patient department of the Hospital for the Ruptured and Crippled more cases of acute anterior poliomyelitis are seen each year than in all the other institutions of New York. At this hospital, during the year October 1905 to September 1906, 379 cases of anterior poliomyelitis and infantile paralysis were diagnosed, while during the following twelve months there were 550 such diagnoses; 272 of the latter cases occurred during August and September. These figures appear to clearly show that during August and September of the present year acute anterior poliomyelitis has been very prevalent in New York.

It is interesting to note that the past summer in New York has been cool and extremely dry. Among the 150 cases examined by Dr Collins or his assistant at the Hospital for the Ruptured and Crippled since August, in only 14 cases was there a history of marked digestive disturbance; nor was it possible, although in every case this point was inquired into, to make out any relationship to defective sanitary surroundings. No special district of the city has been selected. In four instances more than one member of the family was affected; in one instance three children, in the other three, two. The majority of the children were in good health when they were seized by the poliomyelitis.

One of the most striking clinical features of the epidemic is the percentage of cases which make a fairly good recovery. Thus in a considerable number of cases the muscles do not begin to atrophy, but gradually regain their function. Another interesting point is the relatively large number of cases in which the medulla and pons are the seat of the lesion.

The author concludes by indicating that such an epidemic affords exceptional opportunities for examining cases in the first few days of illness, at a period when observations may be expected to throw light upon the pathology of the disease.

EDWIN BRAMWELL.

TWO CASES OF DISSEMINATED SCLEROSIS, WITH AUTOPSY.

(16) J. A. ORMEROD, *Brain*, Oct. 1907, p. 337.

THE first of these cases was of interest because the disease was not suspected during life. There were few or no facts suggesting it. Both cases are reported very fully. The summary of the first is as follows: Woman of 54, onset sudden, ten months before admission.

Extensive spasms of legs, with diarrhoea, repeated on four successive days. Further cramps in legs five weeks later, with pain, finally assuming the form of flexor spasms, lasting for some months. Gradual development of flexor contracture of the lower limbs. Painful tingling in legs six weeks after onset; incontinence of urine with anæsthesia of bladder. Rigidity of abdominal muscles two months before admission. Pain in back and lower limbs. On admission, lower limbs firmly flexed at hip and knee; much tenderness to touch or manipulation; incontinence of urine. Legs straightened under chloroform two and a half months after admission; death from exhaustion nine days later.

The localisation of the disease found post-mortem did not adequately explain the clinical features. There were plaques of disseminated sclerosis in the spinal cord, but principally in the cervical region, and the descending degeneration of the pyramidal tracts was very slight. There were also patches in the pons, medulla, right optic thalamus, and optic tract.

The second case was remarkable for two things. Firstly, the extremely close interweaving of hysterical symptoms with those of disseminated sclerosis. A woman of 38, at one time with an extensive hemianæsthesia and completely bedridden, able after a few weeks' treatment to get about the ward. Babinski's sign and optic atrophy were, however, constantly present. Secondly, a peculiar rash of the skin, coming and going over a considerable period. It began as raised papules, which increased in size and formed either bullæ, small sores, or oval excoriations of the skin. The rash only occurred on anæsthetic skin. Its nature is left in doubt, possible explanations being (1) the expression of an organic nerve lesion, (2) hysterical, (3) manufactured by the patient—this view is favoured but it could not be proved, (4) nutritional change in anæsthetised skin.

There is a good photograph of the position and form of the rash, and diagram of the distribution of the patches of sclerosis.

J. H. HARVEY PIRIE.

THE SACRAL TYPE OF DISSEMINATED SCLEROSIS. (Zur (17) sakralen Form der Sclerosis multiplex.) H. OPPENHEIM, *Neurol. Centralbl.*, Dec. 1, 1907, p. 1106.

OPPENHEIM describes briefly a case which he regards as an example of disseminated sclerosis, the disease manifesting itself in symptoms pointing to disease of the sacral portion of the spinal cord.

The patient, a man aged 46, had been suddenly seized twelve years previously, with retention of urine and weakness in the legs. Rapid improvement followed, though incontinence of urine re-

mained. In January 1906, he had another attack of retention. In June of the same year severe pains in the legs, and later incontinence of fæces, which had been previously present in slight degree, developed. For two years he had been impotent. He had had no cerebral disturbance with the exception of slight giddiness.

On examination, there was no distinct loss of power and no anæsthesia of the lower limbs. The superficial and deep anal reflexes were absent, and there was slight impairment of the perception of cutaneous tactile and painful stimuli in the region around the anus. The left tendo-Achillis jerk was absent, the knee-jerks were very active, the abdominal reflexes were absent, the Babinski sign was present on both sides.

The diagnosis lay between spinal syphilis and multiple sclerosis. No history of syphilis was obtained.

From a study, not only of this case, but of "half-a-dozen similar cases" which he has met with, and the after history of some of which it has been possible to trace (although no details are given in the present short communication), Oppenheim is of opinion that it is permissible to describe a sacral type of disseminated sclerosis.

EDWIN BRAMWELL.

ACUTE MULTIPLE SCLEROSIS OR DISSEMINATED MYELITIS.

(18) E. STADELMANN und M. LEWANDOWSKY. *Neurol. Centralbl.*, Nov. 1, 1907, p. 1001.

A SERVANT girl, aged 26, on August 12, 1906, suddenly complained of pain in the head and, a few days later, of double vision, inequality of the pupils, and a tight feeling round the waist. In the course of the next four weeks vision became very indistinct, and she became unable to walk. On September 15th it was found that she had double optic neuritis; ptosis on the left side; a dilated left pupil immobile to light; inability to count fingers; some weakness of upper extremities; spastic paraplegia with almost complete loss of power in the lower extremities; increased tendon jerks and the Babinski sign; marked diminution in the perception of all forms of stimuli below the third rib; loss of the abdominal reflexes; incomplete paralysis of the bladder and rectum; increased cerebrospinal pressure with marked increase of the cellular elements. A fortnight later there was distinct atrophy of the optic discs; nystagmus on looking to the left; paræsthesiæ in both arms and legs; moderate ataxia in the upper extremities, but no intention tremor. On September 19th patient was somnolent, cystitis was present, and the temperature raised. Death occurred two days later.

On microscopic examination great numbers of patches were found throughout both the brain and spinal cord. In the cerebrum these were situated chiefly at the junction of the grey and white matter. The patches, which were all alike in structure, consisted entirely of glial elements. Even with Bielschowsky's method no stained elements were to be seen in the axis cylinders. Although a widespread secondary degeneration was met with in the myelin sheaths, there was no well-defined degeneration confined to special tracts such as is seen when the axis cylinders are completely destroyed. The authors suggest that the axis cylinders were probably preserved although their presence was not demonstrable.

Finkelnburg and others have urged that the persistence of the axis cylinders is a distinctive feature which characterises acute disseminated sclerosis as opposed to acute disseminated myelitis. The authors regard their case as one of acute disseminated sclerosis.

EDWIN BRAMWELL.

DISSEMINATED SCLEROSIS OR CEREBROSPINAL SYPHILIS.

(19) (Multiple Sklerose oder Lues cerebrospinalis.) KUCKRO,
Münch. Med. Wchn., Nov. 5, 1907, p. 2238.

THE difficulty in distinguishing between some cases of disseminated sclerosis and cerebro-spinal syphilis has been recently emphasised by E. Müller, Erb, Curschmann, and v. Vordt. The therapeutic test is not necessarily decisive, for it may happen that one of these striking periods of improvement, which are well known to occur in the former disease, may coincide with the administration of antisyphilitic treatment. The author describes the case of a young man, aged 30, who eight years previously had had syphilis, which had been thoroughly treated. Paresis and ataxia in the lower limbs with "bladder trouble" developed. These symptoms came on suddenly with severe headache and giddiness. On examination, the condition was as follows:—ataxic paraplegia; increased tendon jerks with the Babinski sign; absence of the abdominal reflexes; normal pupils; right-sided hemihypæsthesia with ataxia of the right hand and diminution of taste, smell and hearing on the right side, pallor of the temporal halves of both papillæ, a central scotoma for colour on the right side. There was no increase of lymphocytes or albumen in the cerebro-spinal fluid. Under potassium iodide and mercury considerable improvement took place. Emphasis is laid upon the central colour scotoma, the absence of the abdominal reflexes, and of a lymphocytosis in the cerebro-spinal fluid as points in favour of disseminated sclerosis, the diagnosis to which the author inclines.

EDWIN BRAMWELL.

ON THE UNION OF TABES DORSALIS WITH DISEASES OF THE

(20) **HEART AND VESSELS.** (Ueber die Vereinigung der Tabes dorsalis mit Erkrankungen des Herzens und der Gefäße.)

STRÜMPPELL, *Deut. med. Wochenschr.*, Nov. 21, 1907.

As late as 1885 the writer remarks von Leyden in an article on tabes, referred to the frequent coincidence of aortic disease with tabes, but denied any intrinsic relationship between them. Different statistics are quoted giving the prevalence of aortic insufficiency in tabes as 1 in 12 to 1 in 7.

He concludes that in patients with aortic incompetence or atheroma, one often finds on special search the signs of oncoming tabes, and in a considerable proportion of cases both diseases are equally far advanced. This is explained by both having the same essential cause, viz. syphilis, which can be found with certainty in at least 62 to 72 per cent. of the cases of the two diseases. He holds the absence of pupillary reaction to light stimulus to be of special value in diagnosing a syphilitic stigma, and so arriving at the cause of aortic disease which may be present.

JOHN D. COMRIE.

THE SYNCHRONOUS MOVEMENTS OF THE LOWER EYELIDS

(21) **WITH THE TONGUE AND LOWER JAW OBSERVED IN CERTAIN DISEASES.** H. J. ROBSON, *Lancet*, Dec. 14, 1907, p. 1681.

THE author has observed slight eversion of the lower eyelids on protrusion of the tongue or depression of the lower jaw to be present in various acute diseases, and also in chronic cases where there is marked asthenia or long-standing pain. It is, he says, never present in health, and may be a clear index or sign of disease. The explanation of the phenomena is not very clear.

J. H. HARVEY PIRIE.

ON THE URINARY CONSTITUENTS IN THE HEMICRANIAS.

(22) (Sul ricambio urinario nelle emicranie.) M. A. BIOGLIO (of Rome), *Riv. Sper. di Fren.*, Vol. xxxiii., Fasc. 1.

In every form of hemicrania there are alterations in the elimination of the principal organic and inorganic elements of the urine. The nitrogen metabolism is slightly retarded in the hemicranias during the intervals between the attacks; the amount of chlorides,

total sulphuric acid, and earthy phosphates is below the normal; elimination of phosphoric acid is normal. During the attack the nitrogen elimination is constantly accelerated; the other elements may or may not vary. There is a marked difference between the epileptic and the hemicranic with regard to metabolism.

C. MACFIE CAMPBELL.

**A CASE OF APHASIA BOTH "MOTOR" AND "SENSORY," WITH
(23) INTEGRITY OF THE LEFT THIRD FRONTAL CONVOLUTION: LESION IN THE LENTICULAR ZONE AND INFERIOR LONGITUDINAL FASCICULUS.** DERCUM, *Journ. of Nerv. and Mental Dis.*, Nov. 1907.

THE writer gives a brief résumé of the views lately advanced by Pierre Marie on the subject of aphasia, and states his belief that much of the previous writing upon aphasia was deduced simply from complicated diagrams and schemes, and was found to be at variance with observed facts. He records in detail a case which forms a parallel to one of Marie, and expresses his conviction that the function of the lenticular nucleus is the co-ordination of complex muscular movements—among others, those of speech. There are references to the various papers of Marie and others dealing with the subject, but the article does not lend itself to be shortly abstracted.

JOHN D. COMRIE.

ANEURISMS OF THE LARGER CEREBRAL ARTERIES. C. F.
(24) BEADLES, *Brain*, Oct. 1907, p. 285.

THIS article is based on a study of 555 recorded or inspected *true* aneurisms of the cerebral arteries, and resolves itself into the symptomatology. Is it possible, from the symptoms produced, to form a diagnosis of aneurism, and of its situation, when occurring within the cranial cavity? The author's answer is, in brief—No, that it is quite impossible to diagnose an aneurism of any one of the cerebral arteries except in the most unusual circumstances. Only two or three have ever been diagnosed during life, and even in these cases it was scarcely certain. He goes further, and says that in the vast majority of cases of aneurism a tumour even cannot be diagnosed. If there is any one sign to which special attention might be drawn, it is the occasional intermittent character of the symptoms. But these form a small proportion even of those cases where tumour symptoms were present. He classifies them into four groups: (1) Those where the first indication is an apoplectic attack from rupture of the sac—46·3 %.

(2) Those in which a fatal apoplexy has been preceded by symptoms suggesting a cerebral tumour or other lesion—20·7 %. (3) Those in which there have been indications of a cerebral tumour only—16·39 %. (4) Those causing no symptoms during life—16·61 %.

Notes of symptoms of a considerable number of cases are given. It may be noted that slowly progressive signs suggestive of a tumour, with subsequent apoplectic seizure, are by no means diagnostic of aneurism. The statement, copied from book to book, that there is often a murmur, audible to both patient and auscultator, should have received here its deathblow. There are only two cases proved after death to be uncomplicated true aneurism, in which a murmur was heard during life. One of these was an aneurism of the vertebral, the other of the cavernous portion of the internal carotid. There are none on record in proved cases of aneurism of the true intracranial portion of the carotid, or on any of the other larger arteries at the base of the brain. The paper is illustrated by a considerable number of plates and figures, and there is a bibliography.

J. H. HARVEY PIRIE.

EPILEPTIFORM CONVULSIONS AND HEMIPLEGIA IN TYPHOID

(25) **FEVER.** (*Convulsions épileptiformes et hémiplégie au cours d'une fièvre typhoïde.*) BARIÉ et LIAN, *Bull. et Mém. de la Soc. méd. des Hôp. de Paris*, Oct. 31, 1907, p. 1080. LAIGNEL-LAVASTINE, *ibid.*, Nov. 15, 1907, p. 1217.

A GIRL, aged 19, whose mother suffered from epileptic fits, had two attacks of Jacksonian epilepsy, followed by left hemiplegia on the fifteenth day of typhoid fever. The paralysis was already much attenuated at the end of twenty-four hours, and had completely disappeared within a week. Hemiplegia with or without aphasia is the rarest form of typhoidal paralysis. Only 34 cases have hitherto been recorded. In this case the date of the onset does not correspond to that noted in most of the cases. Landouzy and most of the other writers agree that the paralyzes of typhoid are most common in convalescence, whereas in the present case the attack occurred during the height of the disease. In discussing the pathogeny of the phenomena the writers exclude hysteria, owing to the presence of Babinski's sign on the hemiplegic side. Cerebral hæmorrhage is also excluded by the absence of coma and the rapid retrogression of the motor phenomena. Meningeal hæmorrhage is negatived by the fact that lumbar puncture did not reveal blood in the cerebro-spinal fluid. In cerebral softening due to embolism or thrombosis restoration of function would not

have been observed so soon. Uræmia could not be regarded as the cause, since there were no other signs of uræmia, *e.g.*, œdema, gallop rhythm, dyspnoea, coma, or gastro-intestinal disturbance. Serous meningitis was set aside by the absence of characteristic clinical signs and by the condition of the cerebro-spinal fluid, which was normal when first examined, and only showed a few leucocytes four days after the appearance of the nervous phenomena. The cause of the convulsive and paralytic symptoms was probably a slight and transitory alteration of the cortical cells by the typhoid toxins. In the child of an epileptic parent the nervous system constituted a *locus minoris resistentiæ*, and so was specially liable to be affected.

Laignel-Lavastine's case was that of a medical student, age 28, who was free from any nervous taint. After a moderately severe attack of typhoid, and shortly before the temperature had reached normal, left hemiplegia, with ankle clonus but no Babinski's sign, developed. The paralysis lasted a week. The pathogeny was probably the same as in the previous case. J. D. ROLLESTON.

GLUTEAL HERPES FOLLOWING LUMBAR PUNCTURE. (*Zona de*
(26) *la fesse consécutif à la ponction lombaire.*) ACHARD, *Bull. et*
Mém. de la Soc. méd. des Hôp. de Paris, 1907, p. 1330.

A WOMAN, aged 23, who was suspected to have syphilis, had lumbar puncture performed. Clear fluid without leucocytes was withdrawn. Five days later she complained of pain in the lumbar region, and a herpetic eruption was found four inches in length, extending obliquely downwards and outwards from the lower end of the sacrum. The eruption, which was associated with a little swelling of the glands in the corresponding groin, soon healed.

J. D. ROLLESTON.

HERPES IN EPIDEMIC CEREBRO-SPINAL MENINGITIS. (*Ueber*
(27) *Herpes bei meningitis cerebrospinalis epidemica.*) EINHORN,
Wien. klin. Woch., No. 23, 1907, p. 700.

HERPES occurring in epidemic cerebro-spinal meningitis is remarkable for its extensive distribution, atypical localisation, and relatively long duration. The eruption may cover the lips, cheeks, ears, neck, and eyelids, and has even been known to extend from the scalp to the nipple. Its atypical localisation is illustrated by the fact that it often attacks the front and back of the pinna, the eyelids, the thumbs, or the scalp. The eruption comes out in crops, fresh vesicles coexisting with scabs, whereas in most other infectious diseases the eruption all appears at once. It usually

occurs from the third to the sixth day of disease. It was present in about half of Einhorn's cases, in about a quarter of which it affected the mucosa. The distribution of the eruption on the mucous membranes is as manifold as on the skin. The hard palate and gums of the upper jaw are most frequently affected, but the soft palate, uvula, conjunctiva, and nasal mucous membrane may also be involved. The pathogeny is still obscure. Drigalski alone has found the meningococcus in the vesicles. There are no complications nor sequelæ. The eruption has no prognostic significance, and requires no special treatment.

J. D. ROLLESTON.

CLINICAL AND BACTERIOLOGICAL REMARKS ON EPIDEMIC

(28) **CEREBRO-SPINAL MENINGITIS.** (*Klinische und bakteriologische Bemerkungen zur epidemischen Genickstarre im Anschluss an drei sporadische Fälle.*) BENNECKE, *Muench. med. Wochenschr.*, Oct. 29, 1907.

THE writer discusses several important features of the disease in reference to three cases which he records very fully. Two of these, in young men, were not fatal, but the diagnosis was established through lumbar puncture. In both cases the meningococcus was found to grow quite as well upon ordinary agar as upon serum agar. The first symptoms of illness in one case followed within some hours of a severe box upon the ear, which the writer is disposed to regard as of a causal nature. In the second case an unusual point was that the spinal fluid was withdrawn at a pressure of 520 mm. of mercury. In the third case, which was that of a woman of 50, and was fatal, the meningococci failed to grow upon ordinary agar, but developed vigorously on serum agar. In this case they were obtained also from the circulating blood.

The writer gives a copious list of references to papers of the past two years dealing with the bacteriology of cerebro-spinal meningitis.

JOHN D. COMRIE.

POLYNUCLEOSIS OF THE CEREBRO-SPINAL FLUID IN THREE

(29) **CASES OF TUBERCULAR MENINGITIS.** (*Polynucléose rachidienne dans trois cas de méningite tuberculeuse.*) LANDOWSKI and CLARET, *Arch. Gén. de Méd.*, Aug. 1907.

THE observations of three cases of tubercular meningitis in which the cellular content of the cerebro-spinal fluid consisted almost entirely of polymorphonuclear elements. In one case a series of punctures showed that the polynucleosis passed into an almost pure lymphocytosis.

C. MACFIE CAMPBELL.

KERNIG'S SIGN. (A Propos du Signe de Kernig.) CHARLES MON-
(30) CANY, *Gaz. des Hôp.*, Dec. 10, 1907, p. 1684.

THE author believes that Kernig's sign depends on rigidity more or less pronounced of the spinal column. Rigidity of the neck and spine, which may be unaccompanied by contracture specially affecting the lower limbs, was present in almost all the cases he examined. Again, the sign is present in all cases of spinal rigidity with lumbar lordosis, Pott's disease, spinal ankylosis, rheumatic contractures in the muscles, and simple lumbago. Flexion of the extended limb to a right angle with the trunk is only obtained at the expense of definite bending of the dorso-lumbar region of the spinal cord. The rotation of the pelvis which results diminishes the distance between the origin and insertion of the hamstring muscles. If, however, rigidity of the spine, from whatever cause, prevents this rotation taking place, the distance between the ischium and tibia remains unaltered, with a consequence that Kernig's sign can be elicited. This explanation of the phenomenon, it will be observed, in no way diminishes the practical value of the sign in diagnosis.

EDWIN BRAMWELL.

OTITIC BRAIN ABSCESS. E. B. DENCH, *Amer. Journ. Med. Sc.*,
(31) Nov. 1907, p. 692.

SHORT notes are given of two cases (one of cerebellar abscess and the other an abscess of the inferior frontal convolution), with an analysis of 102 recorded cases of cerebellar and 100 cases of cerebral abscess: the full bibliography of these cases is appended.

CEREBELLAR GROUP.—The route of infection was through the petrous bone in 30 cases, through the lateral sinus in 30, through the mastoid apparently in 4, secondary to a cerebral abscess in 3, uncertain in 35.

Of symptoms, the most prominent and constant was headache (71 cases), seldom localised; vomiting was rarely noted as absent, and was prominent in 54; vertigo occurred in 30, nystagmus in 17, retraction of muscles of neck in 12, stupor in 44, Cheyne Stokes' respiration in 3, cessation of respiration at operation in 6; pulse was slow in 40; temperature subnormal in 26, high in 5. In 10 cases the state of knee-jerks was noted: in 6 the jerk on the side of the abscess was exaggerated, in 1 the jerk on the opposite side was slightly increased, in 3 the jerk was absent. Pupils were "unequal" in 9 cases, the pupil on the side of lesion being more often dilated; strabismus, usually internal, was present

in 9 cases. Optic neuritis was present in 34, absent in 37, not noted in 31 cases. Of the 102 cases, 33 recovered. In 45 cases the abscess was opened behind the lateral sinus, in 11 cases in front of the sinus; in 46 the method of operation was not stated. The method must depend upon the probable route of infection: Dench, however, thinks it is a fairly good rule, unless the surgeon can definitely trace the infection from the lateral sinus, to make an exploratory opening in front of the sinus, where this is possible.

CEREBRAL GROUP.—In 77 cases the abscess followed chronic otitis media, in 20 it followed acute otitis, in 3 cases the duration of the otitis was not stated. The chief route of infection was through the tegmen tympani (40 cases). Of symptoms, the most prominent and constant were headache (77 cases) and vomiting (44 cases). Slow pulse was noted in 37, rapid pulse in 1; temperature normal or subnormal in 20, high in 7; vertigo in 32, coma and stupor in 31, mental dulness in 20, general convulsions in 5, nystagmus in 4, pupillary symptoms in 17, aphasia in 10. Motor paresis, varying from slight motor loss to complete hemiplegia, was present in 17 cases—on the side opposite the lesion in 15, on the side of lesion in 2. Reflexes were abolished in 1, exaggerated in 7. Optic neuritis was present in 32, absent in 20, not noted in 48.

Of the 100 cases, 52 recovered. In 41 cases the abscess was opened through the tegmen (27 recoveries), and in 37 cases through the squamous bone (18 recoveries); method of operation not stated in 22. The best results are obtained by operating primarily along the route of infection (*i.e.* in most cases, through the tegmen), as the risks of secondary meningitis and of hernia are thus reduced to a minimum. If more space is needed, the opening may be enlarged upwards and outwards through the squamous plate.

A. W. MACKINTOSH.

THE DEFINITION OF HYSTERIA. (Quelques mots sur la définition (32) de l'Hystérie.) ED. CLAPARÈDE, *Arch. de Psych.*, Oct. 1907, pp. 169-93.

I. MISUNDERSTANDINGS arise from the different points of view, anatomical, physiological, or psychological, of definers, or from their unconscious imprisonment within the bounds of familiar specialities, or from the reaction of their therapeutic experience on their conception of the disease. The definition of the disease is a problem distinct from the question of its nature. A definition must needs be the final outcome and not a fundamental condition of the study of the neurosis. Babinski enters the cavil that the delimitation of any subject must precede the systematic study;

his demand is only legitimate if the delimitation be accepted as a provisional approximation.

II. Babinski thus defines hysteria: A special mental state made manifest mainly by disorders that can be called primary, and incidentally by disorders that are secondary. The characteristic of the primary disorders is their potentiality of being reproduced with rigorous exactitude in certain subjects, and being made to disappear again by the sole agency of persuasion. The characteristic of the secondary disorders is their intimate dependence on the primary.

Babinski's practical preoccupation is the differential diagnosis of organic and functional disease, and the full sweep he gives to his definition demands the added corollary: "None of the classical diseases of to-day outside of hysteria can be reproduced rigorously by suggestion." The reference to "certain subjects" lacks precision, and the definition has the graver ambiguity of leaving the reader in doubt whether it expresses a theoretical opinion as to the nature of hysterical disorders or whether it simply makes empirical enunciation of a diagnostic sign.

III. Is the exaggerated suggestibility of hysterical patients really a cause of the hysterical manifestations? Could it not by any chance be an effect, a symptom on the same footing as its fellows? Of the psychogenesis in time of exaggerated suggestibility there are three possible hypotheses: (a) It may be a fundamental characteristic immediately dependent on constitutional mental disorder. (b) It may be an exaggerated reaction. If hysterical disorders are the result of exaggerated reactions, the function of suggestibility may itself be one of the reactions exaggerated. For suggestion is a normal mental function, a sort of mental reflex having the same biological value for the mind that mimicry has for the body. (c) It may be nothing but a weakening of the personality resulting from the inhibition of certain groups of images and memories.

As to invoking auto-suggestion, why should a patient auto-suggest to himself some derangement which has no significance for him, and which he could certainly never beget in his mind? How could the idea of actualising a stricture of the œsophagus or a contracture of the arm or a loss of the power of standing erect suggest itself spontaneously to a patient? It is the auto-fabrication of the idea which is so important. Now the disorder considered in itself is always very different from the idea which gave it birth. Vomiting may be the result of disgust inspired by an action or person, not of the idea of vomiting. The form of hysterical disorders does not as a rule correspond to any representation the patient has made in advance, and so a hysterical disorder is not necessarily a product of suggestion or auto-suggestion.

IV. Architecturally, hysteria is built storey upon storey. Various manifestations which are habitually placed on the same plane do not all belong to the same rung of the evolutionary ladder. They are the fruits of the same tree, but have not ripened on the same branch. A bird's-eye view of the disease can only be got by rising above the raw facts and leaving the atmosphere of purely clinical observation for that not only of psychology but of general biology.

The search after biological significance lets us know whether a reaction is a purposive act or simply an accidental practice. It permits us to recognise family resemblances and a common origin in reactions of dissimilar aspect, and lets it be seen, finally, in what way a reaction becomes abnormal, by excess, by defect, or by deviation.

The examination of hysterical patients establishes the fact that they oppose a considerable resistance to the remembrance of certain groups of memories which bring pain in their train, and are therefore banished out of consciousness. This inhibition is illuminated by the biological interpretation which represents consciousness taking arms against the invasion of painful images or sensations. The suppression is a defensive reaction, and according as it deals with memories, acts, or regions of the body, so it supplies amnesias, paralyses, or anæsthesias. When the inhibition is total we have a fainting fit, which can be considered as the homologue of the simulation of death so widely spread among animals. Applying the same criterion, we may regard the *globus hystericus* as symbolic of an act of organic defence. In man moral causes may provoke the sensation of physical disgust, with contraction of the pharynx.

Trophic, cutaneous, or vaso-motor manifestations may be explained similarly by reference to the important part the external skin plays in the whole animal kingdom as an organ of protection not only passive but active. Perchance hysterical stigmata are revived ancestral reactions whose utility can only be grasped among practising animals. We still have goose-skin and sudden pallor as evidences of fear: such skin phenomena would appear no less bizarre to us than the blisters of hysteria were they equally rare.

What is the cause of this exaggerated and abnormal reactivity? This is the fundamental question of the constitution of the hysterical subject. The nervous apparatus is one with a tendency to reversion, to atavism, a sort of instability or infantilism facilitating the loosening of the latch which holds in check the rudimentary reflexes. In favour of this view is the infantilism so often a feature of the hysterical mind. So also is the greater frequency of hysteria in women, the female being the more con-

servative sex. The play propensities are easily understood as another expression of infantile nervous organisation.

For the proper elucidation of hysteria it is necessary to study the hysterical subject in his lucid intervals or after cure. There remains to explain a crowd of accidents like tics, hallucinations, and algias, which can hardly be considered as throw-backs. Whence comes the augmentation of suggestibility and the duplication of personality? They occupy a more recent place in the genealogical tree. The inhibitions of the hysterical mind throw out of action part of the woof of personality. Mental control is lessened; the ideas which enter consciousness translate themselves more quickly into action; the state of suggestibility is set up. The group of ideas banished into subconsciousness may end by taking a predominant share in directing the activities of the subject, so arises a second personality. The persistent duration of hysterical reactions is a possible result of the disarray of the habitual psychological means of expression and control.

J. ROY TANNAHILL.

**A FATAL CASE OF CHOREA ASSOCIATED WITH DOUBLE
(33) OPTIC NEURITIS AND HYPERPYREXIA IN A CHILD
AGED THREE AND A HALF YEARS.** GEORGE CARPENTER, *Lancet*, Nov. 30, 1907, p. 1521.

RHEUMATIC hyperpyrexia is exceedingly rare in children. In the case here reported the illness began with rheumatic fever, was followed in a fortnight by chorea, and in six weeks there was a mitral bruit and rheumatic nodules. Under arsenic the choreiform movements became less, but hyperpyrexia ensued. The necropsy report is:—Slight thickening of mitral valve, Peyer's patches injected, intestines otherwise normal, brain normal.

J. H. HARVEY PIRIE.

**CONTRIBUTION TO THE KNOWLEDGE OF TETANOID STATES
(34) IN CHILDHOOD.** (Zur Kenntnis der tetanoiden Zustände des Kindesalters.) ESCHERICH, *Muench. med. Wochenschr.*, Oct. 15, 1907.

THE writer remarks that the recognition of tetany as a serious neurosis does not date farther back than to 1890. He means by the term "tetanoid state" an overexcitability of the muscles to mechanical and galvanic stimulation, reserving the name of tetany for actual spasm of the muscles or general convulsions. As the result of examination of 328 children in a Viennese crèche during the first six months of life, he found that this tetanoid state rose in frequency from 2 per cent. in the first month to 56·2 per cent.

in the sixth, being most frequently observed in rachitic and artificially fed children. The writer believes in the theory that the various forms of tetany in childhood are referable to degeneration of the parathyroid bodies, and in support of this quotes researches from his clinic, in which hæmorrhages were found in 38 parathyroids out of 89 cases of tetany examined. He has not, however, obtained any satisfactory result from the administration of parathyroidin.

JOHN D. COMRIE.

PAROXYSMAL TACHYCARDIA. E. SCHMOLL, *Amer. Journ. Med. Sc.*, Nov. 1907, p. 662.

THIS paper, based on nine cases, is "an attempt to show that in paroxysmal tachycardia we have to deal, not with a single pathological entity, but with different pathogenic forms."

The cases are divided into four etiological groups:—

(1) Cases occurring in patients with previously damaged heart. Notes of three such cases are given, and it is pointed out that the heart lesion alone does not cause the symptom-complex, which originates only when a new weakening influence (*e.g.*, a febrile attack, strain, over-exertion) interferes with the action of the heart.

(2) Cases occurring in patients affected by dysthyreosis. Paroxysmal tachycardia was also noted in a case of Dercum's disease (*adiposis dolorosa*).

(3) Cases occurring in patients with central nervous lesions. Notes are given of one case, in some respects resembling an atypical case of multiple sclerosis, but believed—in view of age and extensive arterio-sclerosis—to be a case of vascular disease of the central nervous system, the attacks of paroxysmal tachycardia probably being due to vascular disturbance of the medulla oblongata.

(4) Cases of apparently functional character, so-called idiopathic group.

From a study of a series of pulse tracings of an example of the last group and also of the case of Dercum's disease, Schmoll is led to these conclusions:—

(a) We must distinguish two forms of paroxysmal tachycardia: in one the auricular wave is present, in the other it is absent in the tracing of the jugular pulse during the tachycardiac rhythm.

(b) The paroxysmal attack is best explained in both forms by assuming that it is due to a series of extra systoles originating below the auricle, viz., from the bundle of His.

It is interesting to note that paroxysmal and permanent bradycardia, as well as paroxysmal tachycardia, can be referred to a lesion of this bundle.

A. W. MACKINTOSH.

ON MOTOR, SENSORY, AND VASOMOTOR SYMPTOMS DUE
(36) TO CORONARY ARTERIOSCLEROSIS. (Ueber motorische, sensorische, und vasomotorische Symptome verursacht durch Koronarsklerose und sonstige Erkrankungen der linkseitigen Herzhälfte.) SCHMOLL, *Muench. med. Wochenschr.*, Oct. 8, 1907.

THE writer finds these three groups of symptoms present in cases of arteriosclerosis of the coronary vessels not only during anginal attacks, but also in the intervals between them. Of the sensory symptoms the sense of impending death, and that of pain in the chest and down the left arm, are the most notable. The latter, in the writer's experience, may affect any of the segments from the second cervical to the eighth dorsal, although most common in the parts connected with the eighth cervical and the first dorsal. He states that if the pain is referred to the right side it always, in his experience, is associated with disorder of the right ventricle. Between attacks hyperalgesia is very commonly found in the affected areas. Among the motor symptoms the feeling of constriction (due to spasm of the intercostal and pectoral muscles) and paralysis, causing feebleness of the left arm and inability to hold objects, are the most common. Between attacks, weakness and disproportionate smallness in size of the left arm are common, while some cases have been recorded of actual paralysis and reaction of degeneration in the muscles. The vasomotor symptoms sometimes precede an attack, and then consist, as the writer finds, of irritative nervous phenomena, such as pallor of the fingers of the left hand; but at other times, in persons liable to attack, there is a tendency to vaso-dilatation.

The writer explains all these phenomena as referred somatic symptoms from segments of the cord, disordered by constant afferent impulses by the sympathetic fibres from the diseased heart. He mentions incidentally that aortic diseases are extraordinarily common in San Francisco. JOHN D. COMRIE.

PSYCHIATRY.

TWO CASES OF GENERAL PARALYSIS WITH CEREBRAL
(37) SYPHILIS. (Due casi di demenza paralitica con sifilide cerebrale.) F. GIACCHI (of Reggio-Emilia), *Riv. Sper. di Fren.*, Vol. xxxiii, Fasc. 2-3.

THE first case presented, in addition to the histopathological lesions of general paralysis, a patch of gummatous meningitis, and one of

syphilitic meningo-myelitis. In the second case the syphilitic element in the picture consisted of well-marked syphilitic endarteritis (Heubner) most marked in branches of the Sylvian artery. The author does not discuss the question of the specificity of Huebner's endarteritis, and with regard to the first case the topographical distribution of the histopathological changes is unfortunately not given very fully. In neither case were there sufficient clinical grounds for diagnosing more than general paralysis.

C. MACFIE CAMPBELL.

DELIRIUM TREMENS AFTER WITHDRAWAL OF ALCOHOL.

(38) (*Delirium Tremens nach Alkoholentzug*). HOSCH, *Muench. med. Wochenschr.*, Oct. 29, 1907.

A CASE is recorded in which the patient suffered from old standing non-progressive phthisis and was a strong drinker. On admission to hospital his alcohol was completely stopped and an epileptiform seizure appeared next day, and was succeeded by a typical attack of delirium tremens lasting a week. The writer quotes the opinions of numerous eminent authorities upon the point whether withdrawal of alcohol is capable of causing delirium tremens, and comes to the conclusion that it is safer to diminish the alcohol gradually in patients.

JOHN D. COMRIE.

PSYCHOPATHIC INTOXICATION WITH TRANSFORMATION OF

(39) **THE PERSONALITY.** (*Ivresse psychique avec transformation de la personnalité.*) G. G. DE CLÉRAMBAULT, from the Notes of the late Paul Garnier (of Paris). *Ann. Méd.-Psych.*, Sept.-Oct., Nov.-Dec., 1907.

GARNIER separated three forms of psychopathic intoxication, and in his notes was found the description of a modification of one of these forms, with a transformation of personality.

In this article three cases exemplifying the disorder are recorded. The patients were mildly maniacal, had a feeling of well-being, and had a delirium of megalomaniac colouring; they were convinced of their high rank, and this conviction, with elements derived from the past life of the individual, continued during the whole period of intoxication, and tended to recur in future attacks. This disorder may develop suddenly in alcoholic degenerates, and the megalomaniac element is frequently overlooked owing to the preponderance of the hallucinations. Treatment in an asylum is advisable, and is frequently spontaneously sought by the patient.

J. D. RANKIN.

ON THE ETIOLOGICAL RÔLE IN INSANITY OF VARIOUS

- (40) **ALCOHOLIC BEVERAGES.** (Enquête sur l'importance du rôle joué dans l'aliénation mentale par l'alcool et les boissons à base d'alcool contenant des essences.) M. MIRMAN, Directeur de l'Assistance et de l'Hygiène publiques, *Ann. Méd.-Psych.*, Sept.-Oct. 1907.

A STATISTICAL report of the alcoholic insane of both sexes in France, showing the proportion of cases in each county. The report also gives the statistics of the different alcoholic beverages responsible for insanity. The comparison of the report of 1897 with that of 1907 shows an increase of 57 per cent. in the number of the alcoholic insane.

J. D. RANKIN.

- HYSTERIA AND LITIGIOUS INSANITY.** (Hysterie und Querul-
(41) antenwahn.) K. HEILBRONNER (of Utrecht), *Centralbl. f. Nervenh. u. Psych.*, Oct. 15, 1907.

THE author reports the histories of two cases before taking up the general discussion. The first case was a single woman of 43, constitutionally abnormal if not definitely hysterical. On hearing of the engagement of a physician who had treated her for two years, she began to accuse him of sexual misconduct, and brought a lawsuit against him. The court ordered an investigation into her mental condition, and the diagnosis submitted was "hysteria with pathological mendacity." For years she continued to annoy the physician in various ways, and even placarded the town with scandalous accusations. The latter referred not only to her own treatment, but also to the treatment of many other girls, with regard to which she made very elaborate, but absolutely false statements. The condition had lasted for eight years, and there was no evidence of mental enfeeblement.

The second patient was a man, 55 years of age, with a history of a previous attack of astasie-abasie. On quite inadequate grounds, a quarrel over a pet dog, he began an embittered lawsuit, although previously of a good-natured disposition.

After several months of continual strife, in the course of which he carried his claims to the highest authorities, he fell ill and had a delirium, with hallucinations derived from the circle of his morbid ideas. On recovery from the delirium he obstinately maintained the same aggressive attitude for some time, and accused the various authorities of injustice, but was finally persuaded to regard the incident as closed. He continued to maintain a grudge against his adversary, but was able to resume his ordinary routine.

The two cases are well-marked examples of litigious insanity (*Querulantenwahn*), one variety of paranoia, that form of mental disorder in which there is the development of a fixed system of delusions without mental enfeeblement. Kraepelin has very much restricted this group by excluding from it all cases with evidence of deterioration. These latter he has grouped under dementia paranoides. Litigious insanity he considered to be one of the purest types of paranoia. Some authors (Neisser, Siefert, Bonhoeffer) have taken a different view, and insist on the difference between litigious insanity and paranoia as regards the genesis and symptomatology. In paranoia there is a long initial stage with various symptoms, and only after considerable time does the patient arrive at the formulation of his system of delusions, which is a secondary affair, and the content of which is to a certain extent irrelevant to the fundamental process and changeable.

In litigious insanity the central theme is primary, and may be determined from without and not, as in the paranoiac, from within. It forms the centre round which other delusions are elaborated. The fixed idea of injustice causes misinterpretation of the actual facts, while the paranoiac, in his system of delusions, attempts to explain a long series of already morbid phenomena. The paranoiac shows in the early stage a diffuse morbid suspicion and ideas of reference. In litigious insanity a whole system of ideas of reference springs up; this is not diffuse, but is only in relation to the one morbid trend. Paranoia has essentially a chronic progressive course; in litigious insanity the delusional formation is not fatally progressive, but may come to a standstill, and there may be partial recovery with a certain degree of insight.

As to the mechanism in the two cases; in the first case a morbid idea, arising from a hysterical basis, obtained pathological value and was supported by luxuriant fabrications of the same hysterical origin; in the second case, the extent of the morbid ideas had an external origin, and when a hysterical delirium later occurred, it borrowed its colouring from the morbid trend. In the first case the disorder seemed to spring from the original character of the patient; in the second, it seemed in contrast with his previous character. Although the author in this discussion approaches the attitude of Magnan, who holds that slow progressive delusional formation arises on the basis of a degenerate constitution, he emphasises the fact that degeneracy in all cases of litigious insanity is not at all established as yet. It is premature to apply the term degeneracy to the individual predisposition on which the disorder arises. With regard to the whole question of paranoia, the study of the individual constitution and of the early mechanism is very important.

C. MACFIE CAMPBELL.

THE EXPERIENCE OF SEXUAL TRAUMATA AS A FORM OF
(42) INFANTILE SEXUAL ACTIVITY. (*Das Erleiden sexueller*
Traumen als Form infantiler Sexualbetätigung.) K. ABRAHAM
 (of Zürich), *Centralbl. f. Nervenheil. u. Psych.*, Nov. 15, 1907.

AN abnormal psycho-sexual constitution is, according to Freud, the fundamental condition for the development of the neuroses. Children predisposed to hysteria, owing to this condition, react in an abnormal manner to sexual impressions of every kind. A sexual trauma does not cause a neurosis, but may determine the nature of the symptoms. The question which the author attacks is, Why do so many neuropathic and psychopathic individuals give a history of sexual trauma in childhood? He refers to Freud's important demonstrations that sexuality is an important factor in childhood long before puberty, and calls attention to the fact that children react differently to such traumata. One child immediately relates the experience, another child keeps it secret. The reason is, that in many cases the child, through its subconsciousness, is to a certain extent not merely passive in these episodes. Brief references are made to actual cases confirming this point of view, and the psychological mechanism in hysteria and dementia præcox is discussed.

What has been said of sexual traumata is also of wider application. Frequently an accident is due to the subconscious co-operation of the victim without there being a definite purposeful attempt at suicide. In functional disorders after trauma, with the question of compensation under discussion, the frequency of subsequent traumata to reinforce the claims is due to the same mechanism. In one case this was manifested in an interesting manner by the dreams of the patient, which consisted frequently in the receiving of an injury; the author interprets this as being an example of the fulfilment of a wish on the part of the patient, according to Freud's well-known interpretation of dreams.

C. MACFIE CAMPBELL.

RESEARCHES ON INDOXYLURIA IN MENTAL DISEASES.

(43) (*Ricerche sull' indossiluria nei malati di mente.*) *Riv. Sper. di Fren.*, Vol. xxxiii., Fasc. 1.

THE author, using the methods of Obermayer and Maillard, examined the urine of 118 patients for indoxyl, and found in all an excessive amount present; he tries to correlate in some detail the latter with the clinical symptoms. It is difficult to reconcile

such results with the completely negative results obtained by Folin in his careful metabolism experiments (vid. *Am. Journ. Insan.*, 1904), and by Borden in his still more careful investigation of indican in the urine of the insane (vid. *Journ. of Biol. Chem.*, March 1907).

C. MACFIE CAMPBELL.

SECRETION OF THE GASTRIC JUICE IN ITS RELATION TO

(44) **PSYCHOPATHOLOGICAL CONDITIONS.** (*Die Sekretion des Magensaftes und ihre Beziehungen zu psychopathologischen Zustandsbildern.*) MAYR, *Wien. klin. Wochenschr.*, Oct. 17, 1907.

THE writer records briefly the results of over 200 estimations of the hydrochloric acid, pepsin, and rennin present in the gastric juice of some 90 psychiatric patients. The method adopted was to run 15 cc. of fresh cow's milk into the patient's stomach through the nasal catheter and to withdraw the fluid after five minutes.

In mania the hydrochloric acid, pepsin, and rennin were all diminished.

In amentia the acidity is low, the pepsin very low, but the milk always curdled.

In hysteria and similar conditions, the acidity is strikingly high, the rennin slight, and the pepsin very slight in amount.

In chronic paranoia the conditions are similar to the last mentioned.

JOHN D. COMRIE.

ON A CASE OF TRAUMATIC SOFTENING OF THE CORPUS

(45) **CALLOSUM.** (*Su un caso di rammollimento traumatico del corpo calloso.*) V. FORLI (of Rome), *Riv. Sper. di Fren.*, Vol. xxxiii., Fasc. 2-3.

THE case of a man 47 years of age, who after a severe fall passed through a short period of excitement, with transitory paralysis of the seventh and twelfth cranial nerves of one side, into a state of marked dementia, with no neurological symptoms; he was disoriented, did not recognise his relatives, could give no account of himself, was unclean in his habits; he died one month after the injury. The only lesion found post-mortem was a small circumscribed hæmorrhagic softening of the corpus callosum at the level of the anterior extremity of the optic thalami. Apart from this lesion, the result of macroscopic and microscopic examination of

the brain and meninges was negative. Forli considers the case one of post-traumatic dementia, and refuses to correlate the lesion of the corpus callosum with the mental symptoms. A general review of the various opinions held as to the composition and function of the corpus callosum forms part of the article, and a bibliography containing eighty-two references is appended.

C. MACFIE CAMPBELL.

TREATMENT.

CONTRIBUTION TO THE STUDY OF THE ELECTRICAL TREAT-

- (46) **MENT OF FACIAL TIC-DOULOUREUX BY THE INTRODUCTION OF THE SALICYLIC ION.** (Contribution à l'Étude du traitement du Tic-Douloureux de la Face par l'Introduction Électrolytique de l'Ion Salicylique.) RENÉ DESPLATS, *Arch. d'Élect. méd.*, Nov. 25, 1907, p. 867.

THE writer describes three cases of very severe, long-standing, and intractable tic-douloureux of the face which he has treated by Leduc's method of electrolytic introduction of the salicylic ion. The negative pole, carrying a current of 25 m.a., was placed upon a compress of absorbent cotton-wool soaked in a solution of salicylate of soda, and applied to the affected branch of the nerve for periods varying from one-half to a whole hour.

The results were remarkably encouraging, cure being permanent and complete in two cases, and improvement very marked in the third; and the writer would, in similar cases, have no hesitation in again employing this method of treatment.

ALEXANDER BRUCE.

SUBCUTANEOUS INJECTIONS OF AIR AS A MEANS OF

- (47) **RELIEVING CERTAIN PAINFUL MANIFESTATIONS.**

ALFRED S. GUBB, *Brit. Med. Journ.*, Nov. 9, 1907.

THE results obtained by the injection of air under the skin are approximately the same as those produced by similar administrations of oxygen, hydrogen, nitrogen, or carbonic acid gas. The proceeding is simple and perfectly safe, no untoward consequence having been yet recorded, although the treatment must have been employed in some thousands of cases. The air is pumped by a rubber bellows, such as is used in connection with a Pacquelin's cautery, through a glass bulb containing sterilised cotton-wool, and then through a needle introduced into the subcutaneous tissue

over the painful area. A rounded swelling forms at the seat of puncture, and the air passes along nerve and vessel sheaths, so that secondary swellings may appear at a distance. The initial blanching of the skin is soon followed by a pronounced redness which persists for some hours. As soon as the needle is withdrawn the tumour must be thoroughly massaged, and the massage must be repeated daily until all the air has been absorbed. Absorption is not usually complete for several days. The amount of air injected varies with the seat of the pain. The writer instances 200-300 c.cm. for the gluteal region, and 10-30 c.cm. over the thorax, but in some of the cases detailed as much as 2000 c.cm. was employed in the treatment of sciatica. The procedure is applicable to the relief of pain due to all forms of neuralgia and neuritis. The writer has not yet ventured to treat any of the varieties of facial neuralgia by this means, but thinks that there is no reason why it should not be employed for such cases when analgesics have been found to be insufficient. The article concludes with a summary of nine cases in which the treatment yielded almost uniformly satisfactory results. Five of the patients suffered from sciatica, one from pleurisy, one from intercostal neuralgia, one from a painful burn cicatrix, and one from perforating ulcers of the feet associated with severe pain.

HENRY J. DUNBAR.

THE TREATMENT OF ECLAMPSIA. (*Die Behandlung der*
(48) *Eklampsie.*) BUMM, *Deut. med. Wochenschr.*, Nov. 24, 1907.

THE writer declares that in the very severe forms of eclampsia, numbering 2 to 3 per cent. of all the cases, we are still almost powerless to render successful treatment. He gives the syndrome of symptoms of decreased renal efficiency which in his opinion demand conclusion of pregnancy. Subcutaneous injection of normal saline solution in large quantities three times daily he strongly recommends, and also hot baths or packs. He does not advocate any drugs as beneficial in this condition.

JOHN D. COMRIE.

THE VALUE OF SYSTEMATIC LUMBAR PUNCTURE IN THE
(49) **TREATMENT OF CEREBRO-SPINAL MENINGITIS.** (*Der*
Wert der systematischen Lumbal-punktion in der Behandlung
der Cerebro-Spinal Meningitis.) VON BÓKAY, *Deut. med.*
Wochenschr., Nov. 21, 1907.

THE writer quotes the opinion of various authorities for and against the value of lumbar puncture as a curative measure in

cerebro-spinal meningitis. Among others Franca has employed the method of injecting into the spinal canal 3 to 9 c.cm. of a 1 per cent. lysol solution. Out of 17 cases treated by the writer, by means of repeated lumbar puncture, 10 recovered, of whom one was totally deaf and one partially so. He believes that the case should be punctured at intervals of 1, 2, or 3 days according to the intensity of the disease, but that in children not more than 30 c.cm. should be removed at one time. If the fluid is distinctly purulent or no fluid can be obtained, the use of any treatment is questionable.

JOHN D. COMRIE.

THE TREATMENT OF ARTERIO-SCLEROTIC ATROPHY OF THE
 (50) **CEREBRUM.** (Die Behandlung der arteriosklerotischen
 Atrophie des Grosshirns.) CRAMER, *Deut. med. Wochenschr.*,
 Nov. 21, 1907.

IN a clinical lecture the writer describes the types and treatment of this condition. The early symptoms of importance are headaches, giddiness, and increasing loss of memory; and when these show themselves treatment is urgently demanded. The callings which he finds most liable to this condition are those of publicans, actors, officers, bankers, members of parliament and of boards generally. In treatment he recommends immediate withdrawal of the person from his calling and its attendant excitements, avoidance of alcohol in all forms, removal from the diet of articles which cause flatulence or which are difficult to digest, cessation of violent forms of exercise, and above all regular movement of the bowels. The drugs which he has employed are iodide of potash or of soda, or iodipin, and with these he sometimes combines bromides in equal amount.

JOHN D. COMRIE.

A SURVEY OF THE NEURALGIAS AND THEIR TREATMENT.
 (51) A. EULENBURG (Berlin), *Folia Therapeutica*, Oct. 1907, p. 104.

A BRIEF paper laying stress on the fact that treatment should be causal and not merely anti-neuralgic, although relief of the pain is frequently also imperative. Surgical measures, such as excision of scars, neuromata, etc., are referred to. Specific causes, such as malaria, influenza, syphilis, readily yield to appropriate treatment when recognised. For the frequently occurring neuralgias in anæmic persons he recommends iron and arsenic, particularly in their combination with albumen, marketed as *Arsenoferratose*. In neuralgia in persons of gouty or rheumatic diathesis little need be

expected from colchicum and similar drugs. Regulation of diet and whole manner of living is usually required.

When the cause seems to be some local or general circulatory disturbance, suitable hygienic and therapeutic measures should be adopted; these are very varied. Frequently the cause is not discovered, or it may be beyond direct treatment (inoperable tumours *e.g.*). Special anti-neuralgic remedies and the various forms of physical treatment are to be considered subsequently.

J. H. HARVEY PIRIE.

**NOTE ON TREATMENT OF TRIGEMINAL NEURALGIA BY
(52) INJECTION OF OSMIC ACID INTO THE GASSERIAN
GANGLION.** G. A. WRIGHT, *Lancet*, Dec. 7, 1907, p. 1603.

PROFESSOR WRIGHT, who has already reported the use of osmic acid injection into nerves, has now extended the use of this method for destruction of the Gasserian ganglion as an operation which is much less severe than that required for its removal. Two cases are here reported briefly where the results have been very satisfactory so far, but the time is much too short to warrant a conclusion that the result is a permanent success.

J. H. HARVEY PIRIE.

THE VARIOUS FORMS OF PSYCHOTHERAPY. (La Psycho-
(53) *thérapie dans ses différents modes.*) A. W. VAN REN-
TERGHEM. Amsterdam: Van Rossen, 1907, p. 184.

IN this book the author publishes his communication to the International Congress held at Amsterdam in 1907. The larger part of the book is occupied with reports of cases of various functional neuroses. He emphasises the importance of hypnotism, and does not agree with Dejerine and Dubois in their criticisms of this form of suggestive treatment.

J. D. RANKIN.

Reviews

LES CENTRES NERVEUX CÉRÉBRO - SPINAUX. A. VAN GEHUCHTEN. Louvain : A. Uystpruyst-Dieudonné. 1908. Pp. 469, with 337 Illustrations. Price 25 f.

VAN GEHUCHTEN, who is the author of the well-known text-book, "Leçons sur l'anatomie du système nerveux," has recognised that the anatomy of the nervous system, as it is generally taught to students, or as it is treated in the larger text-books, may not be suited to the needs of the physician who desires an acquaintance with the structure of the brain and spinal cord merely for practical application in the study of the symptoms which arise from diseases in them. The author has consequently attempted, and in our opinion with marked success, to supply the want with this volume, which may serve as a guide to the construction of the nervous mechanisms as far as we at present understand them. His aim has been to present the fundamental facts in as concise a form as possible, excluding those which are merely of morphological or scientific interest, and making only cursory reference to points which are not yet definitely settled.

But in addition to dealing purely and simply with the anatomy of the nervous system, he discusses the physiology and the functions of each part after having described its structure and connections, and he has, wherever it has appeared necessary, dealt with the more essential points of its affection by disease. The pages which are devoted to the physiology and pathology of the neurone are most instructive.

The volume is divided into three parts: the first is devoted to the macroscopical anatomy or morphology of the nervous system; in the second the histology, physiology, and pathology of nerve cells and fibres are first considered, and then the minute anatomy of each part of the nervous system is described with the aid of excellent illustrations and diagrams. The practical manner in which the subject is treated may be illustrated by reference to the chapter on the mid-brain. When the anatomy of this part has been described, the next section is devoted to the consideration of the mid-brain as a special organ, the chief function of which is the innervation of the ocular muscles; here the mechanism for the conjugate movements of the eyes and the pupillary reflex arcs are discussed, and the significance of the disturbance of the function of each is referred to. In the second section the mid-brain is regarded as one of the higher nervous centres and the seat of complex reflex functions, while in the third section it is considered

as a part of the brain-stem through which the tracts connecting the fore-brain and thalamencephalon with the metencephalon and spinal cord must pass.

In the third part the different bundles and systems of fibres which connect the various centres of the nervous system are described and their functions discussed.

The volume is illustrated by 337 figures, including many diagrams, which will allow the reader to follow the anatomical descriptions with as much ease as possible. It is, perhaps, unfortunate that authorities are not cited and that no index is provided; but the book will undoubtedly prove of great value, not merely to students and practitioners who desire only a practical knowledge of the anatomy of the nervous system, but also to neurologists who wish to become acquainted with the more important results of the work of recent years.

GORDON HOLMES.

**BERICHT UEBER DIE LEISTUNGEN AUF DEM GEBIETE DER
ANATOMIE DES CENTRALNERVENSYSTEMS. Dritter
Bericht, 1905 und 1906. L. EDINGER und A. WALLENBERG.
S. Hirzel, Leipzig, 4 M.**

In this report on the contributions to the anatomy and histology of the nervous system which appeared during the years 1905 and 1906, 711 papers are tabulated, and critically abstracted and reviewed.

The report is arranged on the same lines as its now well-known predecessors, and it must be regarded as indispensable to all who are interested in the anatomy or physiology of the nervous system.

The most striking feature of the published work of the last two years is the very large number of papers which have appeared on the finer histology of nerve cells and fibres: over 300 are cited in this review. This has been evidently due to improvements in our technique, and especially to the more general use of Cajal's and Bielschowsky's stains for neurofibrils, and to the fresh impetus these have given to the still raging controversy on the neurone theory. The critical collation of these numerous papers which the report contains must prove of general interest and value to those who have not the time or opportunity to read the original publications.

The section which is devoted to recent modifications or improvements of the methods of examining the nervous system is evidently complete and excellently arranged. The section on the cerebral cortex was entrusted to Brodmann, while Kappers is

responsible for part of that on the comparative anatomy of the brain.

The report extends to nearly 240 pages.

GORDON HOLMES.

THE PSYCHOLOGY AND THERAPY OF NEUROTIC SYMPTOMS.

(Zur Psychologie und Therapie neurotischer Symptome. Eine Studie auf Grund der Neurosenlehre Freuds.) ARTHUR MUTHMANN. Halle: Marhold, 1907, S. 115.

THIS interesting and remarkable book makes its appearance at an opportune moment, when Freud's work is arousing so warm a controversy in Germany. The reviewer is at once faced with the difficulty of discussing the value of the book without being involved in a discussion of Freud's work—a task which is so huge as to be impossible to attempt within the necessary limits of a review. Comment will therefore be confined to considering the merits of the book itself, and the extent to which its objects have been fulfilled. From this point of view one can have nothing but the highest praise, and we congratulate Dr Muthmann on having given us a valuable and moderately worded contribution to the present debate. The object he has had in view appears to be a twofold one: first, to give an adequate sketch of the present standpoint of Freud's theory, and of the recent criticisms and extensions thereof; and, secondly, to bring in support of it fresh evidence based on his personal experience with Freud's method.

As regards the first aim, this has been carried out in a singularly objective manner. Although the author is a whole-hearted supporter of Freud, he chiefly confines himself to giving an impersonal account of the subject, and contributes few fresh criticisms or arguments. The most valuable of his original contributions is, perhaps, that concerning Ricklin's recent criticism of Freud's standpoint on the function of conversion, and in the reviewer's opinion Muthmann's remarks on this point are incontrovertible. This first part of the book was to be considered chiefly, then, as an exposition of Freud's theory rather than as a further contribution thereto. In this the author has succeeded admirably, and in spite of its elimentariness this book is at present the most complete and precise exposition of the present position of psycho-analysis that we possess outside of Freud's writings. The only adverse criticism we have to make in this connection is the inadequate account given of Freud's work on dream interpretation, which is amongst his most original and striking contributions to clinical psychology.

The second and longest part of the book comprises a detailed

account of three psycho-analyses, together with shorter accounts of several others. As the author justly remarks, the question as to the truth or falsity of Freud's theory does not differ from any other problem of natural science, in the fact that it can be solved by only *one* method—the test of experience. He therefore considers that no adverse criticism and no support of the theory is of any value that is not based on actual experience and laborious work, so that the method he adopts of furthering our knowledge of the problems is to relate personally observed facts, and to allow them to speak for themselves. Only by an accumulated mass of such original additions to our knowledge can the various problems at issue be solved. From this empiric standpoint no scientific man can dissent.

In conclusion, we can heartily recommend this volume to anyone who wishes to become acquainted with the new science of psycho-analysis as represented by Freud. Although incomplete, it presents advantages over Freud's writings in being written in a more acceptable and intelligible style, in being less open to misinterpretation, and, in spite of occasional obscure passages, in being worded in a German that is more easily comprehensible to a foreigner.

ERNEST JONES.

DAS FREUD'SCHE IDEOGENITÄTSMOMENT UND SEINE BEDEUTUNG IM MANISCH-DEPRESSIVEN IRRESEIN KRAEPELIN'S. (Freud's Psychogenic Principle and its Importance in Manic-Depressive Insanity.) O. GROSS (of Graz). Leipzig: Vogel. 1907. Pp. 50. M. 1.20.

THE methods of psychological analysis, by means of which Freud has contributed so much to the knowledge of the psycho-neuroses, have proved fertile in the whole field of general psycho-pathology. The Vienna professor succeeded in certain cases in tracing back mental and bodily symptoms to distressing emotional episodes in the distant past, with regard to which there had been an inadequate mental adjustment on the part of the individual. He analysed the process by which such mental elements, submerged and not digested, cause disorders in the conscious life, the origin of which disorders is often unknown to the patient, and is only discovered by the physician after careful analysis. In attributing a dynamic value to such psychological elements, Freud does not profess to have exhausted the etiological factors at work. The disorder often arises only on the basis of a peculiar constitution, such as the hysterical constitution.

In his work, "The Psychology of Dementia Præcox" (vide *Rev. of*

Neur. and Psych., May 1907, pp. 411-20), Jung of Zürich has shown that in this disorder, as in the psycho-neuroses, the nature of the symptoms is to a large extent determined by underlying trends and exiled mental elements, which withdraw energy from the conscious, purposeful life of the individual, and distort his thought and emotions. The dynamic relation of the deterioration to constitution and toxic agents is outside of the scope of Jung's psychological work.

In the present work Gross now examines the bearing of the above principles on a quite different group of cases, those, namely, with the constitutional tendency to a reaction of the manic-depressive type. After some general considerations, in which he insists that the ideogenous nature of certain mental disorders renders the principles of general pathology inadequate in mental pathology, he passes to the somewhat fragmentary, but lengthy, report of a case. The patient was a woman with a history of a series of attacks of depression and of excitement, who had kleptomania in its most classical form, a typical uncontrollable impulse.

The analysis of the latter phenomenon led the author to consider that the morbid impulse to steal was simply the distorted expression of certain elements in the sexual life of the patient. The patient, therefore, was a case of manic-depressive insanity, who at the same time presented the typical symptoms of an obsession-neurosis (Freud). The author discusses in general terms the mutual relationship of the mechanisms involved in the two disorders, which he does not consider to be merely casually associated in this case.

He takes for the basis of his argument Anton's general principle of the compensatory activity of the unaffected parts in lesions of the central nervous system. While a compensatory equilibrium may thus be established, the reserve power of the nervous system is thereby reduced, and symptoms reappear on slighter provocation than with an intact nervous system; thus the symptoms of a focal brain lesion, of a hereditary anomaly, of a psychic trauma, which have been concealed by the compensation, may be elicited during a condition of intoxication.

Similarly, to return to the special subject under discussion, the latent disposition to an endogenous disease, such as manic-depressive insanity, may become patent when the reserve power has been reduced by an ideogenous (psychogenic) disorder; and conversely, the latent ideogenous disorder may first manifest itself during the course of an endogenous disorder, which has reduced the store of compensatory energy of the system.

The author next discusses the part which the ideogenous factor plays inside of manic-depressive insanity itself. In addition to

the two typical phases of this disorder, the manic phase with elation, motor excitement and flight of ideas, the depressed phase with depression, motor retardation, and sluggishness of thought, there are mixed conditions, phases where the symptoms of the two triads are mixed, *e.g.*, a depression with motor excitement and flight of ideas. While the circular mechanism, a constitutional modification of a deep biological principle, may explain the pure conditions, an additional factor must be assumed to explain the dissociation of symptoms seen in the mixed conditions, a dissociation by means of which one of the triad is split off and escapes from the general action of the circular mechanism.

The author concludes with the enumeration of various possible combinations into which enter as elements the individual constitution, the compensatory mechanism, and ideogenous factors. Such expositions are stimulating, and Gross has presented his views very lucidly, but one regrets the absence of well-analysed cases which would form the most solid foundation for such general theories.

C. MACFIE CAMPBELL.

DIE MELANCHOLIE EIN ZUSTANDSBILD DES MANISCH-DEPRESSIVEN IRRESEINS. Eine klinische Studie. GEORGES L. DREYFUS (of Heidelberg). With a Preface by E. KRAEPELIN (of Munich). Jena : Gustav Fischer, 1907. 7 M.

IN this work the author subjects the Melancholia of Kraepelin, the agitated depression of the involution period, to a thorough analysis. On the basis of the cases which Kraepelin himself used, he comes to the conclusion that involutinal melancholia is not entitled to be considered a nosological entity, but that it is merely one clinical picture within the large group of manic-depressive insanity.

The wider issues raised by the conception of manic-depressive insanity are not discussed ; it is an endeavour to carry Kraepelin's own views to their logical outcome. It is therefore rather a discussion within the Kraepelinian school than an endeavour to strengthen the general doctrines of the school by answering the criticisms directed against the formation of the group of manic-depressive insanity.

The author begins with a historical sketch of the process by which the extremely general term melancholia came to receive more and more definition. At first used to include practically all morbid depressions, it was later differentiated into an essential melancholia, and the melancholia which was supposed to be the initial phase of all psychoses. Kahlbaum was the first to ener-

getically protest against this latter being considered as an independent psychosis.

The separation of catatonia by Kahlbaum, of amentia by Meynert, and of circular insanity by the French school, tended to further reduce the heterogeneous group of melancholia; many depressions previously called melancholia were now included in circular insanity.

When Kraepelin, in 1883, brought out the first edition of his text-book, he had not yet advanced beyond the standpoint of his predecessors, and under melancholia we find the heterogeneous varieties melancholia simplex, melancholia attonita, melancholia activa, periodic melancholia—psychoses with different symptomatology and different outcome.

As Kraepelin's breadth of view increased, and when he began to group cases on a wider basis than that of symptom pictures, taking now into consideration mode of onset, symptomatology, outcome, he gradually evolved the conception of manic-depressive insanity. This is essentially a psychosis of a recurrent nature, the various attacks being liable to take either of two forms. The manic phase is characterised by elation, motor restlessness, flight of ideas, while the depressed phase presents a picture of sadness and retardation of thought and action. Besides these two contrasting pictures, there may occur other conditions in which there is a mixture of the symptoms of the two phases.

The great mass of the depressions which were not part of a deteriorating psychosis (*dementia præcox*), or did not belong to definite disorders, such as general paralysis, were now embraced in this large group. Kraepelin refused to include in this group one form of depression, viz. a depression in the involution period, characterised by an anxious agitation, delusions of sinfulness and persecution, and hypochondriacal ideas. The onset of this disorder appeared to be connected with commencing involution; the outcome in the majority of cases was permanent mental enfeeblement. For this group Kraepelin reserved the name melancholia.

Dreyfus having followed Kraepelin so far in his bringing the depressions under the manic-depressive conception, will go farther and maintains the thesis that the agitated depressions of the involution period are merely further varieties of the manic-depressive disorder; and in his preface Kraepelin practically accepts the conclusions of the author.

The grounds on which Dreyfus bases his conclusions are the result of an analysis of the Heidelberg material, but he was able to trace the further history of the patients for more than a decade after Kraepelin had left them. Cases which the latter had described as permanently enfeebled, and which had swelled his percentage of cases resulting in dementia to 49 per cent., were

found to have later completely recovered; and out of 79 patients traced by Dreyfus, only six showed permanent dementia, four of these being undoubted cases of manic-depressive insanity complicated with arterio-sclerosis.

Besides the difference in outcome which the further investigations of Dreyfus showed in these cases, they also disclosed the occurrence of subsequent attacks, sometimes of a manic nature; while a more searching probing of the early history of the patients revealed the frequent occurrence, if not of a previous depression or excitement, at least of characteristic variations of mood.

Not only did these personal inquiries furnish grounds for including the cases under manic-depressive insanity; an analysis of the hospital case-histories elicited characteristic symptoms of manic-depressive insanity. Under this head he mentions the occurrence of transitory elation, irritability, loquacity, flight of ideas, ideas of greatness. The last of these had been wrongly interpreted by Kraepelin to indicate mental deterioration. In view of the fact that the older records had been made without special reference to the present issues, it is probable that such symptoms were much more frequently present than was noted.

In the depressed phase of manic-depressive insanity, which is characterised essentially by sadness and a general blocking of thought and action, the latter may not be shown objectively; it is represented subjectively by a feeling of inadequacy on the part of the patient. The absence of this blocking in melancholia had been insisted on by Kraepelin as a differential point from the manic-depressive depression. Dreyfus admits that it may not be present in a marked form, but considers that it is extremely frequent in the form of a mild subjective feeling of diminution of the intellectual functions, of the emotional responsiveness, of volitional vigour, and in the form of a feeling of fatigue and languor. The author lays great stress on this "partial subjective blocking," which he uses, perhaps, in a somewhat schematic manner.

The material upon which the clinical analysis is based consists of 81 cases, which are presented fully and in an extremely convenient form. The orderly presentation of the histories, with short summaries in tabular form, make the facts easily accessible. This is the first opportunity which one has had to examine the actual clinical material which Kraepelin used in arriving at the views put forth in his text-book. A similar publication of the cases used in the development of the group of dementia præcox would be very acceptable.

The excellent work contained in this book brings out the absolute necessity of careful clinical records, and the advantage of following cases over a long period. The author is to be con-

gratulated on the extremely clear and orderly presentation of the subject, which makes the argument easy to follow.

His point of view may, perhaps, allow a somewhat limited outlook, but this consistent elaboration of the Kraepelinian doctrines clears the way for the discussion of the wider issues involved.

C. MACFIE CAMPBELL.

**DIE ERKENNUNG UND BEHANDLUNG DER MELANCHOLIE
IN DER PRAXIS.** (Diagnosis and Treatment of Melancholia
in Practice.) Prof. TH. ZIEHEN (of Jena). Halle: Marhold.
1907. 2nd Edition, pp. 67. Price 2 M.

IN this edition, as in the first, Professor Ziehen aims more at giving a useful description of the symptomatology and treatment of the cases which he groups as melancholia, rather than at discussing the more general psychiatric issues. In the differential diagnosis he does not discuss the diagnosis between melancholia and the melancholic phase of circular insanity, and in his discussion of periodic melancholia he states that in only one of about thirty cases had a manic attack followed the melancholia. In several cases, however, he had noted what he calls a "reactive" elation after the attack. He gives as the essence of melancholia a primary, unmotivated depression, and a primary retardation of the thought process.

The depression is complicated by anxiety, except in hypomelancholic conditions, and secondary delusions of a depressive nature frequently ensue. The anxiety has frequently a special somatic localisation, and various sensory symptoms are complained of.

The bedside examination of the patient, the important points to note at each visit, the considerations which should determine the advisability of home or hospital treatment, are all clearly gone over. In the treatment Ziehen emphasises the value of opium, which guarantees both a shorter and milder course of the disease; with this many will be inclined to disagree.

The second edition is based on a considerably wider material than the first, and while not treating the subject in its wider aspects, is an excellent practical guide to the physician.

C. MACFIE CAMPBELL.

Review of Neurology and Psychiatry

Original Articles

CLINICAL AND ANATOMICAL DIAGNOSIS OF THE ANKYLOSING DISEASES OF THE SPINAL COLUMN.

By Dr ANDRÉ LÉRI of Paris.

PART II.

II. ANATOMO-PATHOLOGICAL DIAGNOSIS AND PATHOGENESIS.

CLINICAL study reveals differences between these forms of ankylosing diseases which are sufficiently clear to justify their having been separated from each other and to prevent their being re-assimilated. Various writers, however, have refused to separate *spondylose rhizomélisque* and even hereditary traumatic cyphosis from the ill-defined group of so-called rheumatic affections. They base their views mainly on the etiology, which shows that rheumatism and *spondylose rhizomélisque* may be preceded, although with very different degrees of frequency, sometimes by an infection, sometimes by the arthritic diathesis, and even sometimes, in the case of one of these affections, by a more or less severe trauma. I myself have observed a case of *spondylose rhizomélisque* in a man who had had a fracture of the cervical part of the spinal column. Is this a sufficient reason for identifying these processes? Pathological anatomy, as much and even more than clinical observation, shows clearly that they may and ought to be distinctly separated, even if, in virtue undoubtedly of certain common etiological conditions they may, very exceptionally, occur together.

1. *Rheumatism of the Spine*.—Rheumatism of the spine has been very well studied from the anatomical side especially by Professor J. Teissier (of Lyons). The spinal column is either straight or curved to a variable degree. The great anterior common ligament is thickened and ossified throughout the whole length or at one part only, but always irregularly. It is moniliform, much thicker at the level of the intervertebral discs than at the level of the vertebral bodies (Fig. 1). At the level of each disc, and sometimes in the intervals between them, there exists a very marked bony protuberance, either over the whole breadth or over a portion only of the breadth of the disc. This hyper-ossification is especially marked in the lumbar region, and irregular bony protuberances, *osteophytes*, are its most characteristic sign. For this reason Professor Teissier has well proposed to give it the denomination of “vertebral osteophytic rheumatism.”

The protruding bony proliferation is present over every portion of the spine; the vertebral bodies which are not fused together present on their superior and inferior margins a protruding margin of newly-formed bone, which considerably increases their surface (Fig. 2). All the apophyses, spinal, transverse, articular, as well as the laminae are thickened, as if puffed out (*soufflées*), and are more or less fused together by the partial and irregular ossification of their ligaments. The intervertebral discs are partially ossified. As to the intervertebral foramina, they are irregularly narrowed by the thickening of the neighbouring pedicles, or by the presence of osteophytes, which explains the compression of the nerves and spinal roots and the very severe, intercostal neuralgic pains which are frequently noted in spinal rheumatism.

In short, the dominating feature in chronic spinal rheumatism, localised or generalised, is the *protuberance* and the *irregularity* of the osseous new-formations. These encroach upon the bones, upon the cartilages, upon the ligaments, upon all the peri-articular parts. The extreme deformity of the spine strikes one at first sight.

2. *Spondylose Rhizomélisque*.—This is not so in *spondylose rhizomélisque*. A well-prepared spinal column from a case of *spondylose* has at first sight exactly the appearance of a normal column, apart from the general curvature. One is surprised to find, however, that it is completely ankylosed and constitutes only one single bony mass.

The anterior common spinal ligament is almost completely intact, with the exception of some small ossified portions, mainly in the cervical and lumbar regions (Figs. 3 and 8). This ossification is not accompanied by projection. The spinal apophyses, elongated but not swollen, are fused together, especially in the dorsal region, by ossification of the slender band of the interspinous ligament (Fig. 3). The intervertebral discs are completely, or almost completely, free from any ossification (Fig. 5). The articular apophyses are fused together by a perfectly smooth osseous collar, which represents the articular ligaments. After a longer or shorter time we find on section that their spongy tissue has become fused and continuous. The ligamenta sub-flava are especially ossified (Fig. 5). These ligaments and the laminæ which they unite form only one continuous, slightly undulating osseous band. The ribs are united to the vertebræ by direct ossification, partly of the costo-transverse ligaments and partly of the costo-vertebral ligaments (Figs. 6 and 7). These latter ligaments are formed of three bundles, the superior and the inferior, which are strong, uniting the rib to the neighbouring vertebral bodies, and the middle, which is weak, uniting it to the disc. This latter alone is generally slightly, if at all ossified, and forms a depression between the small bony columns formed by the superior and inferior fascicles.

Thus the predominant feature here is the *ossification of the ligaments*, and this ossification takes place *in the substance of the ligament itself, without forming any projection, and in an almost absolutely regular manner*. We may also say that it takes place *fibre by fibre*, an osseous band replacing each fibre of the ligament without the intervention of a cartilaginous phase, for, in the still incompletely ossified yellow ligaments, we could follow the process and see the remaining parts of the ligament of the fibres, becoming directly continuous with true osseous stalactites or stalagmites, coming from the neighbouring vertebral plate (Fig. 9). There is undoubtedly a certain degree of accessory hypertrophy of the articular extremities, since we could note the continuity of substance from one articular apophysis to the other, but always without any abnormal projection.

At the hip joints I have observed a large collar of bone, more or less irregular, corresponding to the capsular ligament and to the completely ossified cotyloid ligament, with conservation of the inter-articular space (Figs. 10, 11, 12, 13).

Thus we have the explanation, as I had previously suggested, of the limitation of the ankyloses, apart from the spinal ankyloses, to the joints of the hips, the shoulders, the knees, and to the sternoclavicular and temporo-maxillary joints. The ankylosis of *spondylose rhizomélique* results essentially from the ossifications of the ligaments. The glenoid and cotyloid ligaments and the menisci are reinforcements of the ligaments—complementary fibro-cartilagenous ligaments. Now the ankylosis affects precisely *all the articulations, and these only, which have a meniscus or an intra-articular fibro-cartilage similar to the cotyloid ligament*; this is because they alone have an ossification of their capsular ligament, sufficient to prevent the movements of the joint. Other joints may be affected by the process, as is proved by the frequent diffusion of the pains at the onset over all the joints; but it is only in very exceptional cases and at a very late stage that they become ankylosed and that their ligaments, which have not the re-inforcing structures mentioned above, become sufficiently ossified to limit the movements.

There remained the further question, by what process an infection such as gonorrhœa, for example, could bring about such a general and systematic ossification of ligaments? We have discovered the cause in our clinical and anatomical observations. Clinically, the constant, regular, and always pronounced curvature of the spine, the very remarkable flattening in every case of the thorax and pelvis, already suggested a *bony softening*. The satisfactory therapeutic results of straightening the spinal curvatures by means either of continuous traction or by the simple action of weight, confirmed this hypothesis. Radiography had revealed a veritable subsidence of the lumbar column into the pelvis, with a tilt of the sacrum, so that the picture of the pelvis, like a heart on a playing-card, recalls that of an osteomalacic pelvis. A woman suffering from spondylose, observed by Ascoli, had in fact been treated first as an osteomalacic. Dr Bécclere, whilst making a radiographic study of one of our cases, has brought to our knowledge the abnormal transparency of the vertebral bodies, of the transverse apophyses, and of the part of the iliac bone corresponding to the fossa. Pathological anatomy confirms all these facts by showing us (Pierre Marie) the extreme *thinness* and abnormal friability of the bones, the diminution of the compact tissue, both near the joints (cotyloid cavity, head of the femur, etc.) and at a distance from them (iliac fossa, transverse apophyses, femur, tibia,

etc.). In one case notably the transverse process could be crushed between the fingers, the head of the femur could be almost cut with a knife, the bottom of the cotyloid cavity and of the iliac fossa were as thin as a sheet of tissue paper. Hilton Fagge, in a case published in 1876, had already noted the extreme friability of the bones.

It is thus clear, from clinical and anatomical facts, that the softening of the bones precedes the ossification of the ligaments which essentially constitutes *spondylose rhizomélisque*. The disease is *primarily a rarifying osteopathy*, a kind of osteomalacia, predominating certainly in the epiphyses, just as the ordinary osteomalacia is limited mainly to the diaphyses. This osteopathy is of infectious origin.

But by what mechanism is the subsequent ossification of the ligaments brought about? Are we dealing with a special and isolated mechanism, or with the application of a general law? In our opinion we have to do solely with an ossification "by *functional adaptation*," and with a simple application of a general mechanism of consolidation, of repair—in short, of recovery from the atrophies or softening of bone. Julius Wolff has studied the transformation of the osseous tissue when it has to sustain pressure or exaggerated traction. His "law of transformation of the bones" is thus formulated. "The exaggeration of pressure and of traction, by reason of trophic stimulation and in the interest of statics, results in the formation of a material which is in a condition to maintain the requisite resistance." Holzknecht, forming his opinion from numerous radiographs of fractures, dislocations and nervous arthropathies, has also explained the abnormal ossifications of connective tissue by the importance of the functional adaptation. "There exist," he says, "in conditions in which pressure has become abnormal new formations of bony substance in the soft parts. These new formations compensate in a crude, and therefore all the more striking way the *impaired equilibrium* of the bones. . . . They supervene when the skeleton can no longer compensate the disturbance by a transformation of its own internal substance. . . . They do not occur accidentally, here and there. They do not appear ever to be primary, but always secondary, and the primary modifications are always coarse destructions of the skeleton, of passive maintenance of the weight of the body, which on one hand markedly diminishes its solidity, and on the other hand produces

this modification." Thus we have the explanation of "the ossification of the capsules and of their reinforcing structures, peri-articular ligaments, tendons, muscles and aponeuroses." This explanation applies absolutely, in our view, to the ossification of the ligaments which, following a primary rarifying osteopathy, constitutes *spondylose rhizomélisque*.

But still further, in the case of the spine itself, we have had numerous proofs that it is indeed by functional adaptation that the ossification of the various ligaments takes place when the skeleton has become insufficient. We have found in very numerous specimens from Pott's disease that, when deep erosion of a vertebral body tends to produce, or has already produced, a forward curvature of the spine, it is not in front, between the vertebral bodies, that the ankylosis usually takes place, but almost always behind, between the vertebral laminæ, *at a distance from the tubercular focus* (Figs. 14, 15, 16, 17). *This ankylosis is due to ossification of the over-strained ligamenta sub-flava*, and is destined to meet the needs of "functional adaptation." In specimens of spinal fractures and dislocations we have found the same consolidation by ossification of the over-strained ligaments (Figs. 18, 19, 20). In marked scolioses we have also observed the ossification of the costo-transverse and costo-vertebral ligaments on the concave side, the side at which they are more strained, and where they sometimes constitute a strong, newly-formed osseous column, which limits the tendency to lateral displacement of the vertebræ (Fig. 21). Even in *spondylose rhizomélisque* the localisation of the ossifications shows that they are formed by functional adaptation. The ossified ligaments, in fact, occupy the convexity of the curvatures; for example, the interspinous ligaments in the cyphotic dorsal region, the most anterior part of the discs, or the anterior common spinal ligament at the level of slight cervical and lumbar lordoses; that is to say, that they could not be better placed for the purpose of *limiting* the deformities and abnormal curvatures of the spine.

Thus we see how essentially different in pathological anatomy, pathogenesis, and clinical appearances are the two great ankylosing diseases of the spinal column.

3. *Hereditary Traumatic Cyphosis*.—The third disease which we have to consider, hereditary traumatic cyphosis, differs quite as much from the two preceding forms.

It is at the *concave* part of the hunch-back that in this case we find

almost the whole of the new osseous formations (Figs. 22 and 23). The main lesion consists in the *moniliform ossification of the great anterior common spinal ligament*, an ossification of a variable and irregular extent, and occupying in length the anterior part of the bodies of 4, 5, or 6 vertebræ. There are many other new bony formations, but very irregular in distribution, in certain portions of the interspinous ligament, for example, or in certain very limited parts of some ligamenta sub-flava; and like those of the anterior common ligament, these ossifications take the form of *projections* and of limited *nodosities* (Fig. 23), either outside the spinal cavity, or in the spinal canal, or in the inter-vertebral foramina.

The anatomical investigation which we have made has given us a very exact idea of the singular evolution of this cyphosis, which suggested to some authors (Kümmel, Henle, etc.)¹ the most varied hypotheses. We have said that the causal trauma is always produced in one of two ways—either by the subject falling on his back, or by the fall of a heavy weight upon his back, the effect of both accidents being to suddenly straighten the normal dorsal cyphosis. The result is a severe laceration or a detachment, with or without involvement of the vertebral periosteum, of the anterior common ligament. These lesions are naturally all the more marked when the normal cyphosis has been already exaggerated by a hereditary or acquired predisposition, and when the suddenly straightened arc of the circle already had a short radius. In the days following the patient suffers pain at the level of the lesion, and the cyphosis becomes accentuated by reason of the pain and the reflex contracture. Then he straightens himself again, frequently to a more erect position than before the accident, precisely because the lacerated anterior ligament no longer contracts the spinal arc as before in its movements of extension. But in the months which follow, repair takes place, and there, as elsewhere, the lacerated ligament, along with the small portions of injured periosteum or cartilage, becomes repaired by more or less exuberant ossification; and it is this ossification itself which produces the more or less marked curvature of the spine. That is why in cyphosis

¹ Kümmel had supposed that the friction of the vertebral bodies by the traumatism might disturb their nutrition to such a degree as to cause the subsequent softening: this hypothesis was admitted by Hattemer and by Kaufmann. Mikulicz and Henle think the formation of a traumatic hæmatoma, inside or outside the dura mater, would cause the softening of the vertebræ by the compression of the spinal roots and ganglia.

we find the new bone formations limited mainly to the concavity of the curvature, and not, as in *spondylose*, to the convexity. Its localisation is such that we can see *a priori* that it must have *produced* the curvature and not limited it. That is the reason also why this projection (*gibbosité*), whether it be permanent or not, cannot be reduced by suspension, because it is the result of an osseous ankylosis, whilst the cyphosis, which may have been pre-existing or not, remains reducible.

We have had, apart from hereditary traumatic cyphosis, other anatomical demonstrations of this pathogenesis of traumatic lesions of the spine. We have, in fact, observed a very similar ossification of the great anterior or spinal ligament, a large moniliform ossification, extending the length of 4 or 5 vertebræ, in a spine in one of the vertebral bodies of which we found a revolver bullet which had been received some years previously. The violent injury had in this case undoubtedly acted in the same way as the traumatisms of cyphotics, by suddenly straightening the spinal curvature and by lacerating or detaching the anterior ligament.

But this, the main lesion of the anterior ligament, is not the only one; isolated or grouped fibres of various ligaments may have been ruptured by the injury. Repair takes place there also by more or less exuberant new-formations of bone. These form the small disseminated nodosities which we have described. When, as we have observed, these nodosities are situated on the internal surface of the ligamenta sub-flava, in the spinal canal, they may compress the cord and produce the more or less serious signs of compression of the cord of which we have spoken.

III. THERAPEUTIC DEDUCTIONS.

We see, in short, that the anatomical diagnosis, as well as the clinical diagnosis of these three ankylosing affections of the spinal column is absolutely distinct. But the knowledge of this anatomy has an advantage quite apart from a purely theoretical interest. It has a practical therapeutic bearing, and that of the first order. Certainly our knowledge of spinal ankyloses is still far from complete, but such as it is, such as we have just described it, it already lends itself to important therapeutic deductions.

Chronic deforming rheumatism of the spinal column will be treated medically in the same way as chronic rheumatism of any other localisation—with salicylates, iodides, etc., according to the



FIG. 1.



FIG. 2.

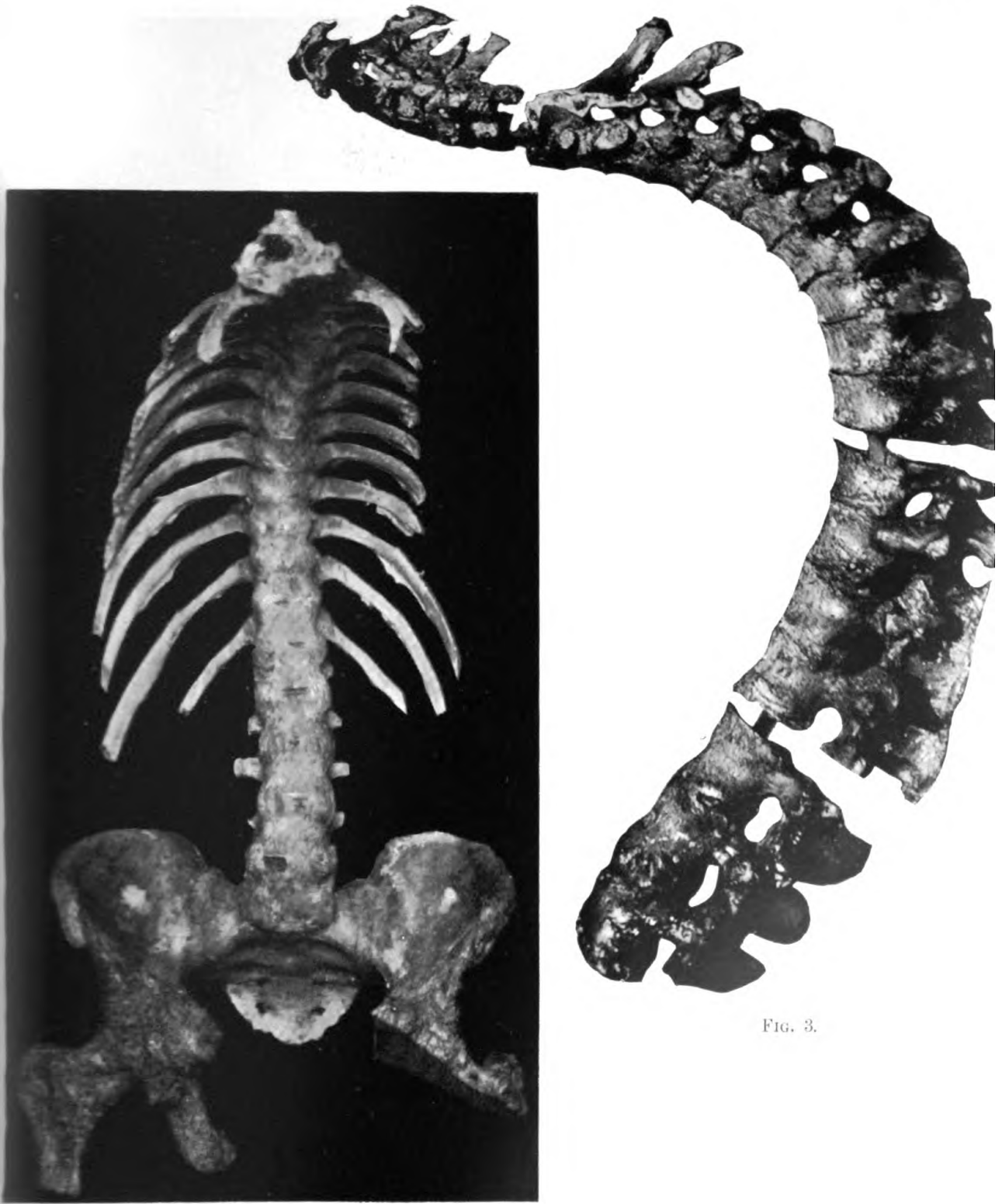


FIG. 4.

FIG. 3.



FIG. 5.



FIG. 6.

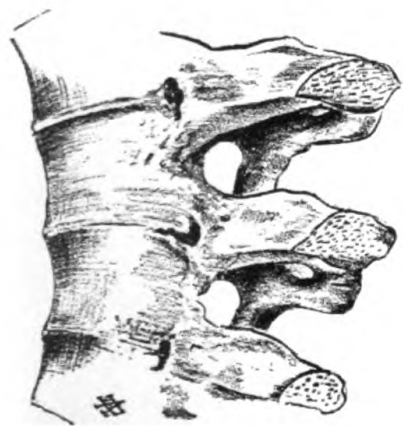


FIG. 7.

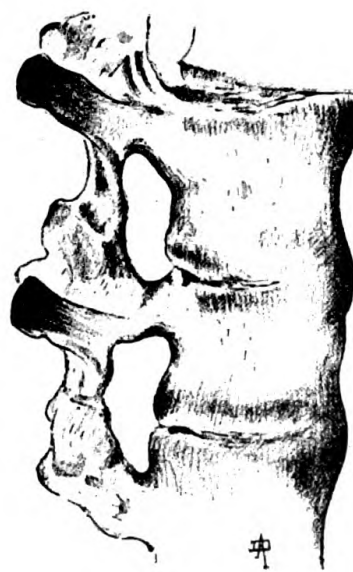


FIG. 8.

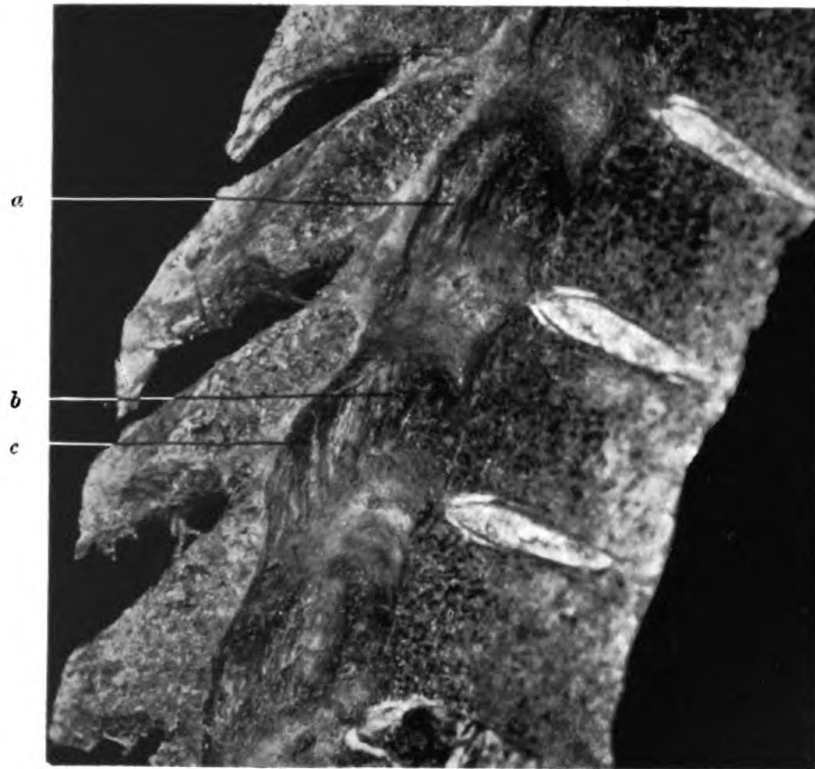


FIG. 9.

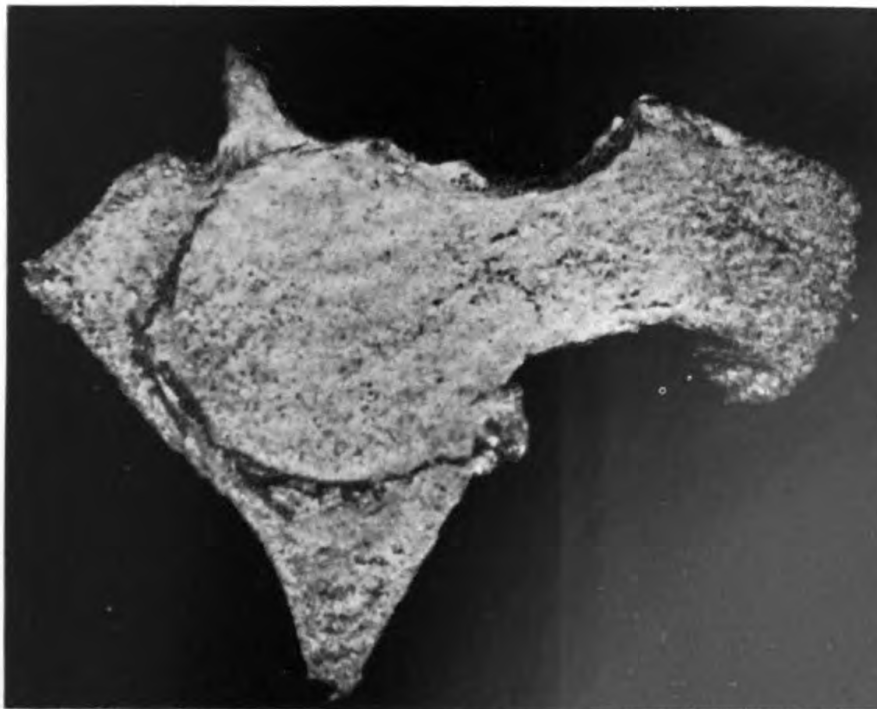
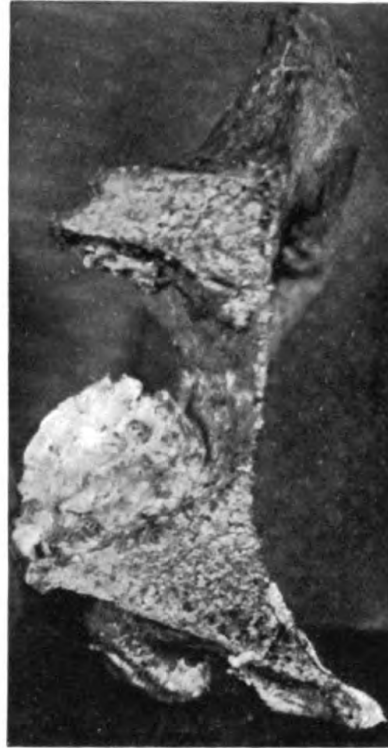


FIG. 10.



FIGS. 11 and 12.

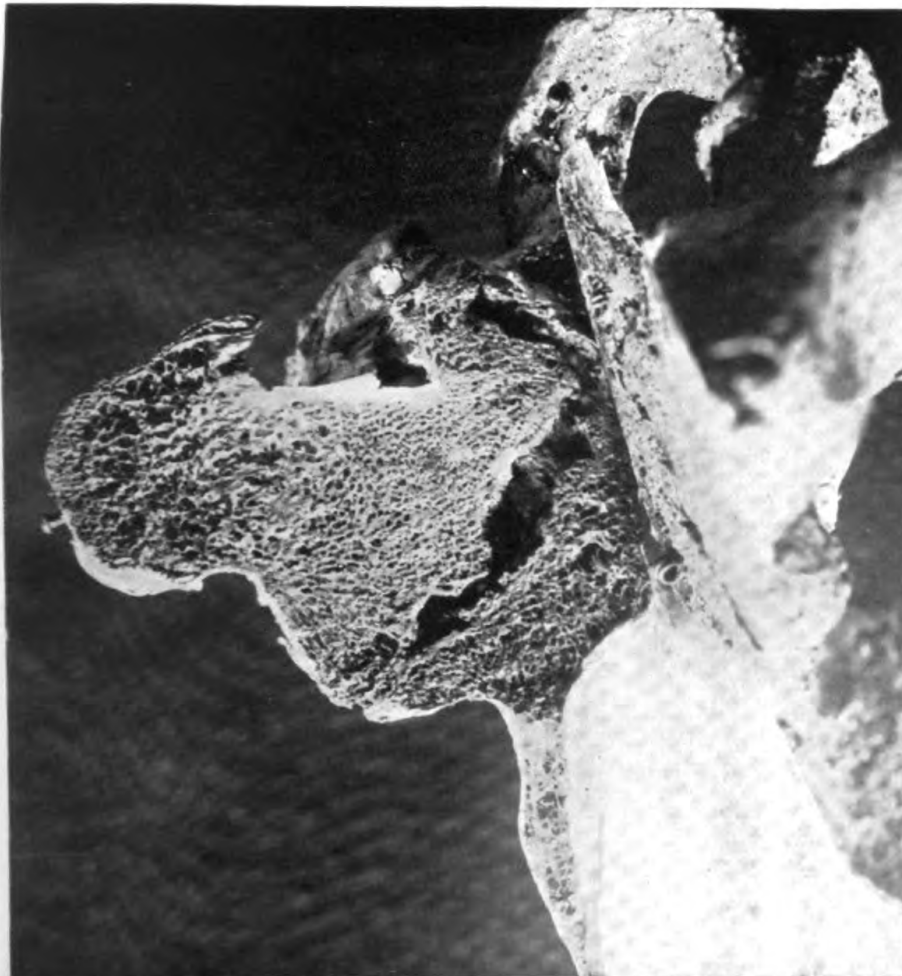


FIG. 13.

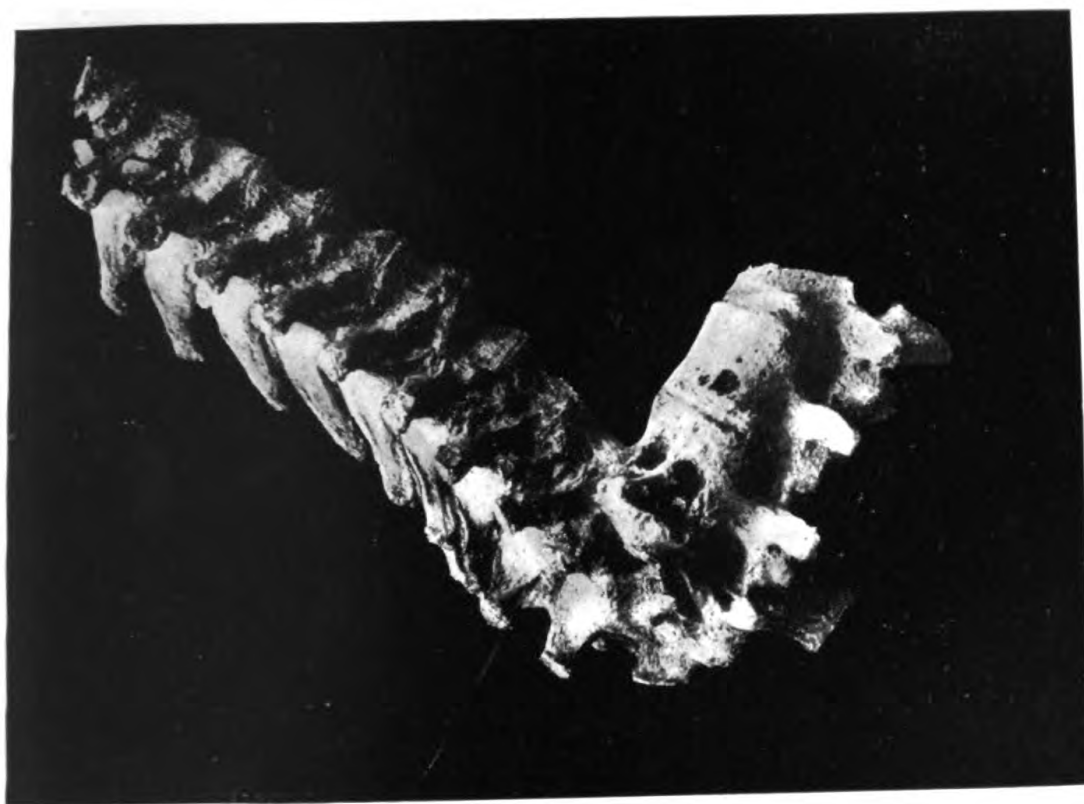


FIG. 14.

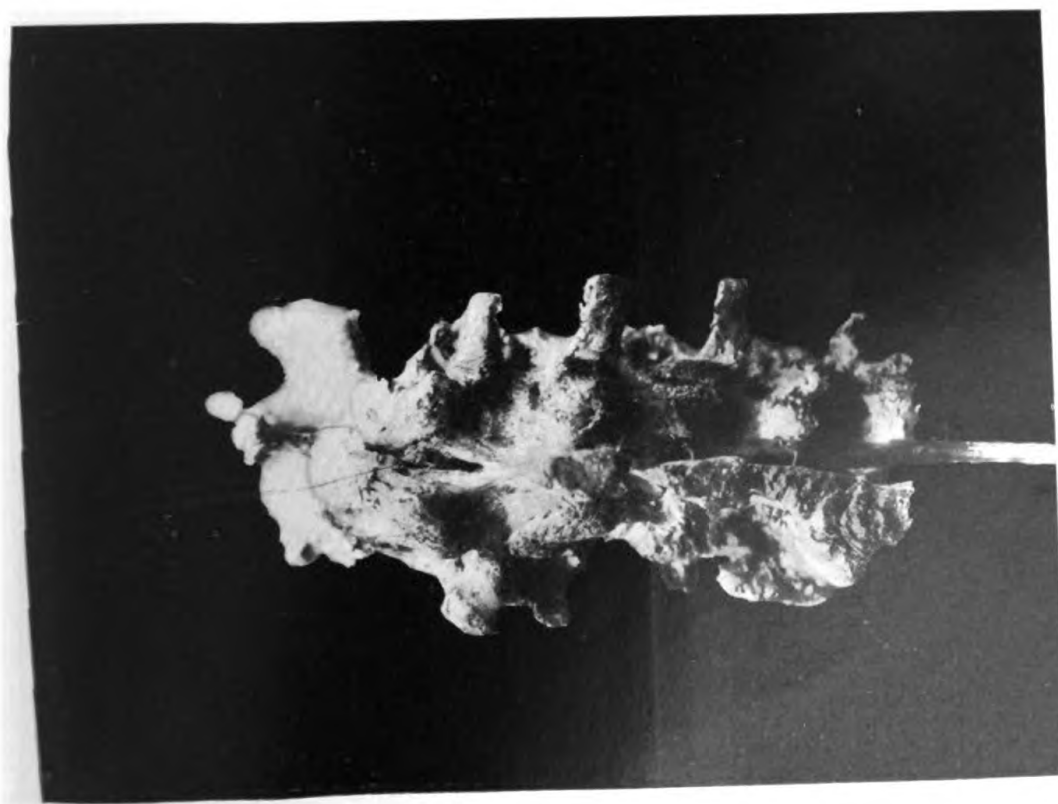


FIG. 15.

1

a
a
a



FIG. 17.

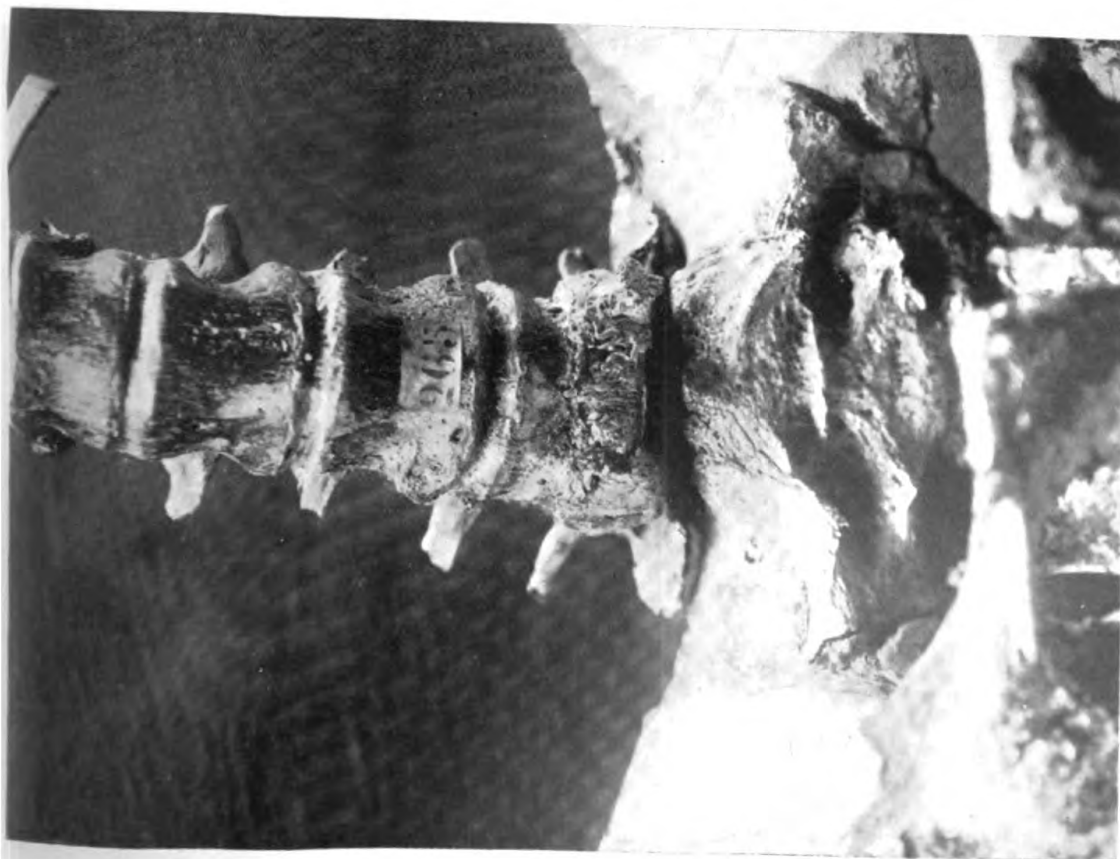
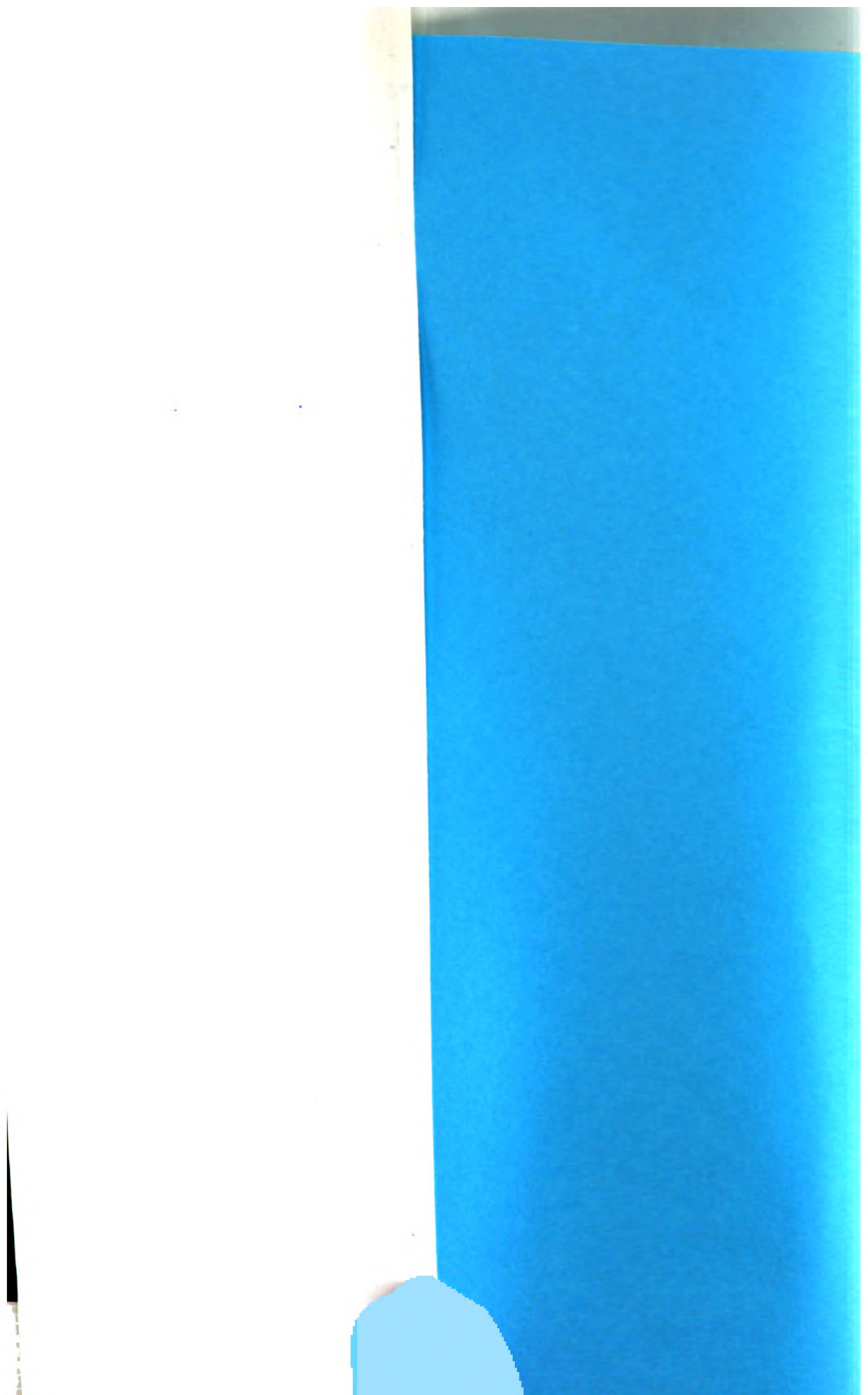
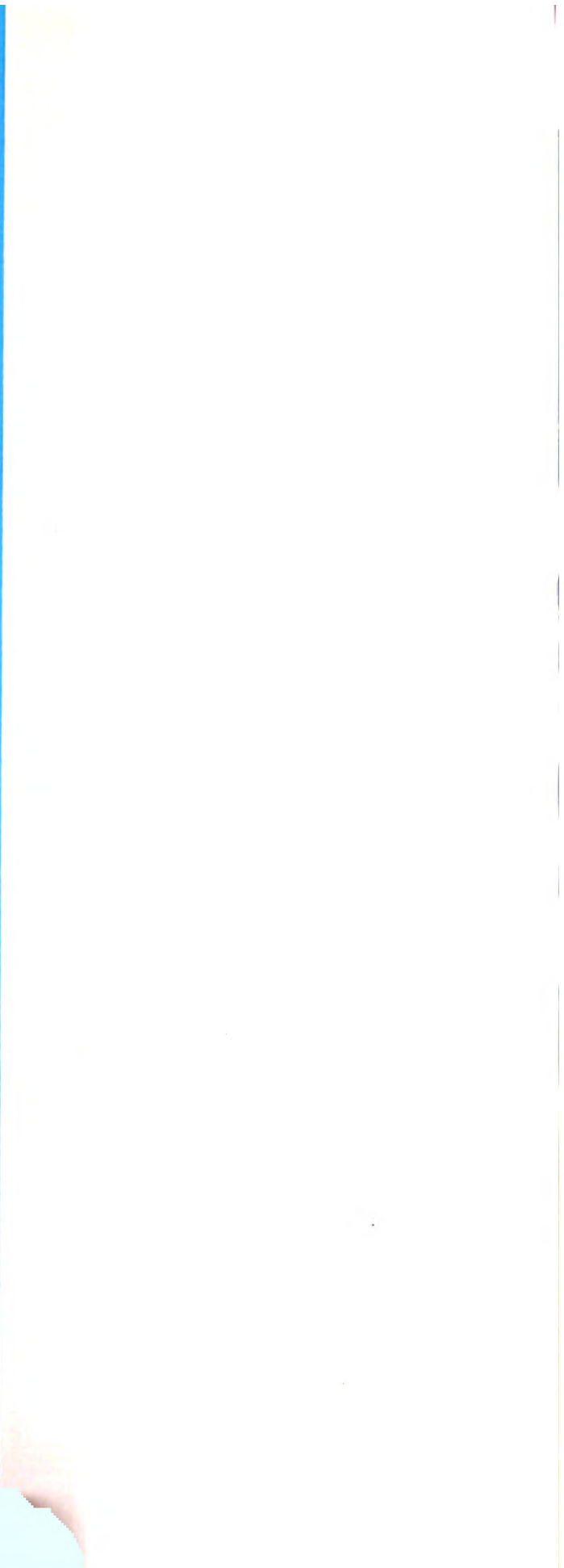
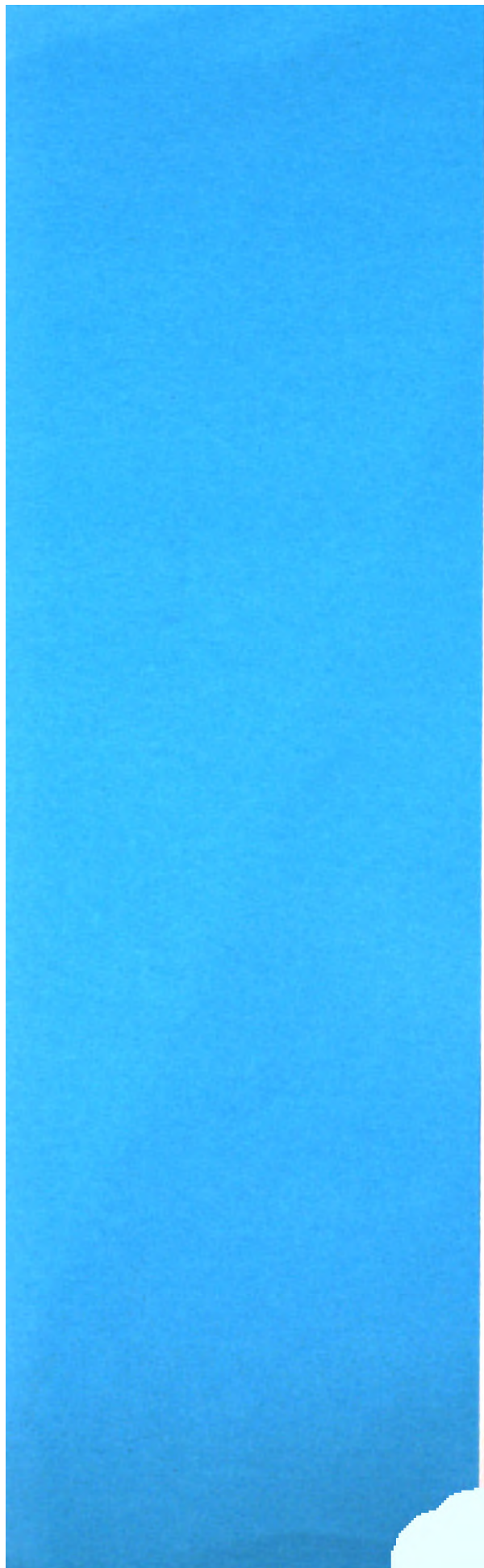


FIG. 16.







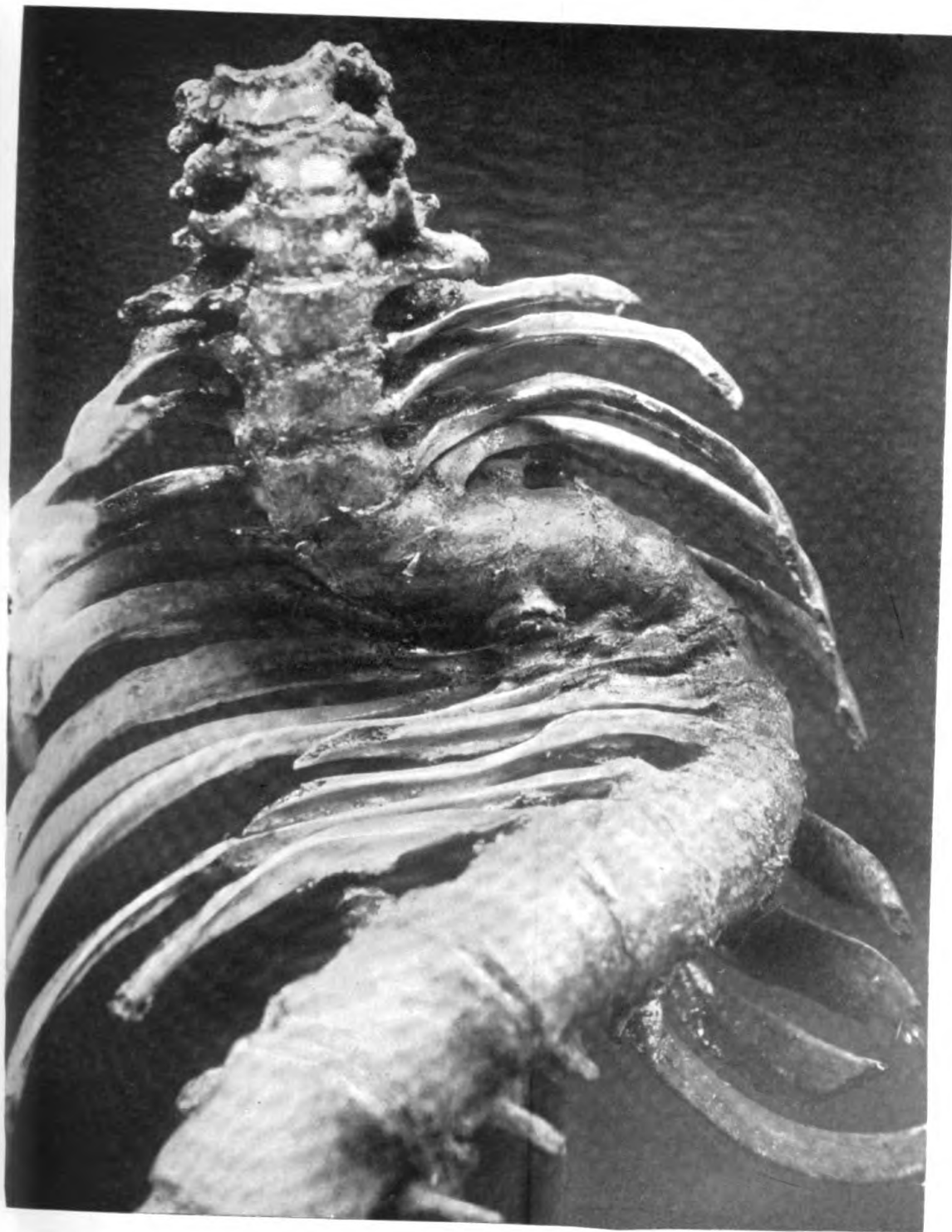
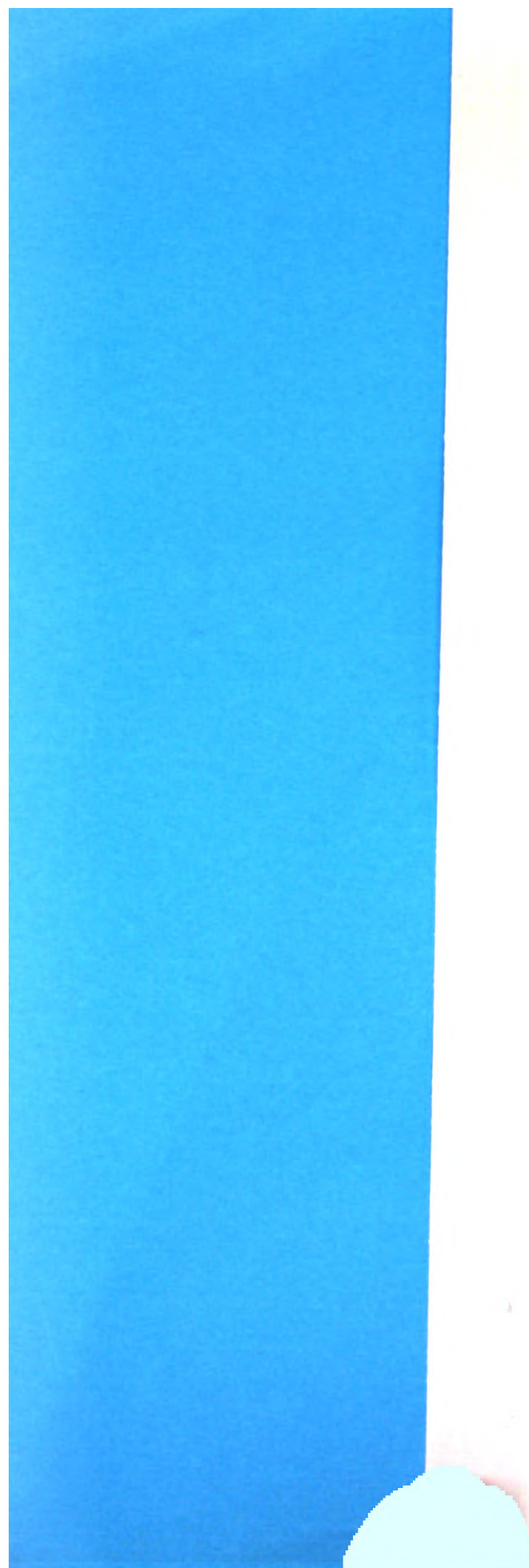


FIG. 21.





case. But if there appear severe intercostal neuralgic pains, we will know that they are caused, in all probability, by a shortening of the intervertebral foramina, and the knowledge of the radicular localisation of the disturbances of sensibility will, in certain particularly persistent cases, allow of a direct attack, by surgical intervention, upon the cause of the pains.

In *spondylose rhizomelique*, some interventions have been made, notably by Dr Nélaton, which aimed at removing the ankylosis of the hip by resection of the head of the femur. These have all failed, and even the interposition of a muscle between the osseous surfaces could not prevent a return of the condition. We can now perfectly understand these failures, since the new ankylosing ossification is not the cause of the disease, but a process of recovery, since this process of recovery is the simple application of a law of general pathology, and since, in default of articular ligaments, the new formations may and must take place in all the peri-articular tissues, connective tissue, tendons, even muscles, as Holzkecht has seen in some cases. In order to attack the joints with any chance of success, it would be necessary to make sure that the "osteomalacic period" of the disease is passed; and as yet we have no criterion by which to make sure of this.

On the other hand, the procedures which are directed not to the hyper-ossification—the secondary process,—but to the bony softening—the primary process—have often yielded remarkable results. We have found, as Bäumlér also did, that the simple action of weight, applied, for example, whilst the neck is supported by a cushion, allowing the head to hang down without a pillow, will straighten a marked forward inclination of the head. We have also found, in the wards of Dr Walther, that continuous traction, applied by means of a chin-strap for the same purpose, may have a rapid and remarkable result. These results may not be permanent, but they may be repeated on several occasions, and in any case will render very great service to the patient. We have also found that somewhat forcible passive movements will restore to a shoulder joint, through rupture of the still slight peri-articular ossifications, a mobility which was very much restricted.

This is not all: the same medical treatment which is of service in infectious chronic rheumatism, by means of salol, for example, may also be of very real service in cases of *spondylose* which have the same infectious etiology. And very frequently it will be possible

to attack it at the source of the infection, which, as we have said, is often more or less latent, but is by no means always inactive. In numerous cases, for instance, the causal gonorrhœa may still be revealed by a slight turbidity of the urine, and repeated washing of the bladder has seemed to us to be in such cases the best therapeutic adjunct, and if not to bring about the retrocession of the ankylosing process, at least to limit it.

In *hereditary traumatic cyphosis* the projection may, as we have said, be more or less completely cured, when the ossification of the anterior ligament is not yet too marked, by rest in bed and the wearing of a corset. Later there may sometimes appear more or less serious signs of compression of nerve roots or spinal cord. It is most important to know that this compression is due to small nodes of bone, and that by searching for the well-known localising signs of lesions of the cord or its roots we may possibly be able, by a timely surgical intervention which is comparatively simple, to procure the disappearance of the intractable neuralgias or the paraplegic symptoms.

The study, especially the anatomical study, of spinal ankylosis is as yet only at its commencement. It seems to us even now to furnish therapeutic indications sufficiently precise and important to deserve in future more numerous investigations, the more especially when we consider that we are dealing with affections against which surgery has hitherto appeared to be absolutely powerless.

DESCRIPTION OF FIGURES.

Fig. 1.—*Spinal Rheumatism*. Note the numerous and irregular osteophytes, the voluminous protuberances, especially at the level of the discs. (*Musée Dupuytren.*)

Fig. 2.—A vertebra, seen from above, in a case of *Spinal Rheumatism*. Note the development of the osteophytes which surround the vertebra and almost double its surface.

Fig. 3.—*Spondylose Rhizomélisque*. Note the general curvature of the spine without any *gibbosité*, the integrity of the anterior spinal ligament apart from some absolutely smooth ossifications in the cervical and lumbar regions, the complete absence of osteophytes, the integrity of the intervertebral space, the ossification of the supraspinal ligament in the dorsal region. (This figure was published in the Report on "The Clinical Forms of Chronic Rheumatism," by Prof. J. Teissier, Congrès de Médecine de Liège, 1905.)

ANKYLOSING DISEASES OF SPINAL COLUMN 75

Fig. 4.—*Spondylose Rhizomélisque*. (In this case there is a fracture of the cervical column in addition to the *spondylose*.) Note the smooth ossification of some parts of the anterior spinal ligament or of the discs in the dorso-lumbar region, the absence of osteophytes, the ankylosis of the ribs to the vertebræ, the subsidence of the spine into the pelvis, the tilt of the sacrum, the complete ankylosis of the hips formed by the ossification of the ligaments surrounding the joint.

(Figs. 5, 6, 7, 8.—Different portions of the spinal column in
Spondylose Rhizomélisque.)

Fig. 5.—*Cervical portion*. Note the complete ossification of the ligamenta sub-flava (the plates and the ligaments forming one continuous osseous band), the integrity of the discs and the absence of any osseous protuberance.

Fig. 6.—*Mid-dorsal portion*. Note the ossification of the costo-vertebral ligaments (especially the superior and inferior fascicles), of the costo-transverse ligaments, and the lengthening of the spinal apophyses.

Fig. 7.—*Mid-dorsal portion*.—Note specially the mode of ossification of the costo-vertebral ligaments.

Fig. 8.—*Lumbar portion*. Note the slight, smooth protuberances at the level of the discs, and the ossification of the inter-articular ligaments.

(These figs. were published in the *Nouv. Icon. de la Salpêtrière*, 1906.)

Fig. 9.—*Spondylose Rhizomélisque*. Lines a, b, and c point to the osseous stalactites at the level of the intervertebral discs in the ligamenta sub-flava.

Fig. 10.—*Spondylose Rhizomélisque*. Oblique section of the hip seen in Fig. 4. Note the conservation of the joint cavity, the integrity of the head of the femur and of the cotyloid cavity, the ossification of the cotyloid ligament and of the other ligaments which has produced the complete ankylosis, the rarefaction of all the bony tissue. (Fig. published in the *Nouv. Icon. de la Salpêtrière*, 1906.)

Figs. 11 and 12.—The hip joint of the preceding fig. divided into two portions. Note the nodes of new-formed osseous tissue protruding greatly upon the iliac bone at the level of the cotyloid ligament which has entirely disappeared, and upon the femur at the point of contact of the node, the marked thinness of the bottom of the cavity, and the great spongiosity of all the tissue except that which is new-formed. (Figs. published in the *Nouv. Icon. de la Salpêtrière*, 1906.)

Fig. 13.—Hip in *rheumatism* (*morbus coxæ*?). In spite of the persistence of the articular inter-line, one is struck by the absolute dissimilarity between this lesion and the lesion of the hip in *spondylose*. (*Musée Dupuytren*.)

Figs. 14 and 15.—*Dorsal Pott's disease*. Distinct angular inflexion of the spinal column from destruction of the vertebral bodies. In Fig. 15 (which represents the inferior part and the apex of the angle seen from behind) we see the simple ossification of the yellow ligaments (which has occurred without any osseous protuberance) between the spinal plates which are not affected by the tubercular process. This ossification could hardly be better placed for the purpose of *limiting* the deformities; it seems to be due to a "functional adaptation" of the tissue of these ligaments. (*Musée Dupuytren*.) (Figs. published in the Report by Dr Léri on the "Pathogeny of the Ankyloses, and especially of the Vertebral Ankyloses," Congrès pour l'Avancement des Sciences, Lyon, 1906.)

Figs. 16 and 17.—*Lumbar Pott's disease*. Destruction of the body of the 5th lumbar vertebra (Fig. 16) has produced an inflexion of the column which is limited behind by the ossification of the ligamenta subflava. This ossification is best seen in Fig. 17 at the points marked a. (*Musée Dupuytren*.)

Fig. 18.—*Fracture of the 2nd lumbar vertebra* by the buffer of a locomotive; death five months later. Note the new-formed osseous band, due to the ossification of the lacerated spinal ligament, perhaps also of the connective tissue which ascends along the 3rd and 2nd lumbar vertebræ. This ossification, produced by the traction, could not be placed better for the purpose of limiting the descent of the superior portion. (*Musée Dupuytren*.) (Fig. published in the Report by Dr Léri on the "Pathogeny of the Ankyloses," Congrès de Lyon, 1906.)

Figs. 19 and 20.—*Dislocation of the 6th cervical vertebra on the 7th*. In Fig. 20 we have the posterior view of the smooth ossification of the substance of the ligamenta sub-flava which limits the displacement. (*Musée Dupuytren*.)

Fig. 21.—*Scoliosis of the 3rd to the 12th dorsal vertebræ*. Note the thick new-formed ossifications of the vertebræ and ribs, which, from the concave side, "line" the spinal column, as one might say, and prevent the forward movement from becoming more marked. The new ossifications are found chiefly (1) on the concave margin of the intervertebral discs, at the point where the disc undergoes the maximum traction and the vertebral bodies the maximum pressure; (2) at the level of the costo-vertebral and costo-transverse ligaments. The latter, oblique from above downwards and from the outside inwards, from the superior rib towards the transverse apophysis, and from the transverse apophysis towards the inferior rib, are particularly exposed to the strain from the concave side of the scoliosis. (*Musée Dupuytren*.)

Fig. 22.—Spinal column in *hereditary traumatic cyphosis* (B), between two columns from *spondylose rhizomélique* (A and C). Note the difference in inclination—gentle and complete in the *spondylose* (column A shows

in addition a cervical fracture), abrupt and almost angular, but very limited in the cyphosis. Also ossification in the latter of the anterior vertebral ligament at the level of the concavity of the column. (Fig. published in the *Nouv. Icon. de la Salpêtrière*, 1906.)

Fig. 23.—*Hereditary traumatic cyphosis*. Median section at the level of the *gibbosité*. Note (1) the dense ossification of the anterior spinal ligament and of the anterior part of the column at the level of the concavity (a); (2) a protruding nodosity in the spinal column (b), formed by an osteophytic ossification isolated by a yellow ligament.

THE EPICONUS SYMPTOM-COMPLEX IN CEREBRO-SPINAL SYPHILIS.

By WILLIAM G. SPILLER, M.D.,

Professor of Neuropathology, and Associate-Professor of Neurology in the University of Pennsylvania; Neurologist to the Philadelphia General Hospital.

Read in abstract before the Pennsylvania State Medical Society, Sept. 1907.

From the Department of Neurology and the Laboratory of Neuropathology of the University of Pennsylvania, and from the Philadelphia General Hospital.

It is not often that the symptoms of an epiconus lesion are caused by syphilis, and yet a case has been observed by the author in which the diagnosis had to be made between syphilitic multiple neuritis and a lesion of the epiconus or its roots. The existence of the former condition is questionable. Remak thinks it is not yet positively determined whether syphilis may cause polyneuritis, although it is probable. Flatau refers to a case studied by Oppenheim and Siemerling in which the saphenous major and the cruralis nerves showed a slight decrease of nerve fibres and a slight increase of the endoneurium. The radial and peroneal nerves were intact. This is the only case with necropsy he mentions, but he states that pathologic-anatomical findings in a case of pure syphilitic polyneuritis have not been obtained. Implication of the cranial nerves is common in syphilis of the brain and is caused by the syphilitic meningitis; a similar involvement of the spinal roots from syphilis of the spinal cord also occurs frequently.

Remak says that Ehrmann has observed cases of neuritis nodosa on a syphilitic basis, and he (Remak) has seen painful swellings of nerves, especially of the ulnar, radial, and peroneal nerves, and swellings of the brachial plexus in cases of localised syphilitic neuritis. In a case of brachial neuritis on a syphilitic basis, studied with Westphal, the nerves were hard to the touch, but Remak remarks that such swellings occur in non-syphilitic cases and cannot always be attributed to the syphilis. In cases in which the neuritis was supposed to be produced by syphilis, there were other signs or history of syphilis, or improvement from anti-syphilitic treatment. Syphilitic neuritis is rare, whereas other manifestations of syphilis are common. A few clinical cases of syphilitic mononeuritis are referred to by Remak.

A brachial neuritis may be simulated by pressure on the plexus by enlarged syphilitic glands. Bilateral symmetrical paralysis may be caused by implication of the vertebræ or meninges, as in a syphilitic case observed by Remak,¹ in which the circumflex nerve was paralysed on each side and the fifth and sixth cervical vertebræ were thickened. Primary bilateral syphilitic brachial neuritis may occur, in Remak's opinion, and he refers to a clinical case of Leyden's as an example. He refers to clinical cases of syphilitic polyneuritis reported by Gross, Oppenheim, Buzzard, Fordyce, Taylor, Schlossberger, Sorrentino, Perrero, Brauer, Spillman and Etienne, Crocq, and Fry.

Some so-called cases of syphilitic neuritis may have been caused by the mercurial treatment. In Brauer's case² the treatment with mercury had been employed, and Bauer was uncertain whether both the mercury and syphilis together caused the polyneuritis, or whether other causes existed. Mercury had been employed five weeks before the symptoms of polyneuritis appeared. Neuritis was found by microscopical examination, and some of the cells of the anterior horn were vacuolated.

Cestan,³ in 1900, reported two clinical cases of syphilitic polyneuritis, and collected eleven cases he found in the literature. In both his cases the neuritis occurred very soon after the infec-

¹ Remak and Flatau, "Neuritis and Polyneuritis, Nothnagel's System."

² Brauer, *Neurol. Cent.*, 1896, p. 671.

³ Cestan, *Nouvelle Iconographie de la Salpêtrière*, 1900, p. 153.

tion. Of the cases he collected, only two were with necropsy (Brauer, Kahler). In regard to mercury being the cause of the polyneuritis, he refers to the fact that Lewin observed only once symptoms of neuritis in 8000 cases of syphilis treated by injections of the bi-chloride. Cestan's two cases were without sensory involvement, and suggested very much the form of neuritis seen in lead palsy, inasmuch as the symptoms were purely motor, and in the first case the paralysis was confined to the upper limbs and was most pronounced in the extensors of the hands. It is possible, I think, that the symptoms in these cases were caused by lesions of the spinal cord and not by peripheral neuritis.

Oppenheim, in the fourth edition of his text-book, p. 537, says that Schultze, Buzzard, and he (Oppenheim) have described cases of syphilitic polyneuritis, and Cestan recently has also reported unquestionable cases. He acknowledges its existence, but speaks of it as a very rare affection.

In the discussion following the report of Fry's case of syphilitic multiple neuritis before the American Neurological Association,¹ Dana, Starr, J. J. Putnam, and Leonard Weber said they had never seen a case of syphilitic multiple neuritis. Starr,² in the first edition of his text-book, published in 1903, expresses himself a little more guardedly, and says there is a certain probability that some of the cases were of syphilitic origin, but the condition is extremely rare. At a discussion of the New York Neurological Society it was found that no one had seen a case of multiple neuritis undoubtedly syphilitic.

The case that forms the subject of this paper is as follows :—

The patient, a coloured man, admitted syphilitic infection. He denied alcoholism, except that he had occasionally taken a little beer. He entered the Philadelphia General Hospital, July 27, 1905. About three months previously pain had been felt in the back on the left side, low down near the os innominatum. Numbness and pain were then felt in the left lower limb, especially severely in the calf. When he entered the hospital he moved the left lower limb in walking as in foot-

¹ Dana and others, *Journal of Nervous and Mental Disease*, vol. xxv., 1898, p. 598.

² Starr, "Organic Nervous Diseases," Lea Bros. & Co., 1903, p. 160.

drop. The power of flexion of the left foot was impaired. No tenderness was felt over the nerve trunks, and the patellar reflexes were preserved. The voluntary movement of all the limbs at this time was good, except in the dorsal flexion of the foot. When the sole of the right foot was irritated, flexion of toes was produced, but irritation of the sole of the left foot caused no response. Ankle clonus was not obtained. The patellar tendon, triceps and biceps tendon, cremasteric, and epigastric reflexes were preserved and equal on the two sides and about normal, indeed, the patellar reflexes seemed a little prompter than normal. Achilles jerks were not obtained.

The pupils were unequal, the right being the larger. Reaction to light was absent, but contraction in convergence was preserved. The extra-ocular muscles were normal. The tongue was not affected. The functions of the bladder and rectum were not disturbed. Sensation, objectively tested, was normal.

Aug. 12, 1905.—An examination by Dr William Pickett on this date revealed that the right pupil was myotic, but the light reflex was obtained, and in a dark room the right pupil became larger. Dorsal flexion of the feet was performed only by the tibialis anticus muscles. Tactile anæsthesia was present on the dorsum, outside of the foot and plantar surface on each side, and on the lower and outer part of the legs, and was more pronounced in the left limb, where it extended nearly to the knee. The man complained of pain at night nearly circling the body at the level of the iliac crests. An area of anæsthesia was found near the arm on the left side, about 10 cm. in breadth.

Sept. 14, 1905.—Difficulty in talking was observed on this date, and speech was unintelligible. The man was weak and drowsy. He understood at times what was said to him, but often failed to understand commands: for example, he raised his upper limb when told to put out his tongue. He had more difficulty in moving his left lower limb. When he was aroused he opened the right eye but kept the left eye closed, or opened it only slightly and with effort. He was able to forcibly close the eyelids. The muscles of the facial nerve supply were not affected. The tongue was protruded straight. The left eyeball was not rotated outward on voluntary movement, and moved very little in convergence, and slightly upward and downward.

The patellar reflex was exaggerated on the right side, on the

left side it was not so prompt, but not diminished. Achilles reflex was absent on each side. The plantar reflexes were preserved.

Oct. 22, 1905.—An examination was made on this date by Dr William J. M'Connell in Dr Mills' service. Ptosis of the left upper lid was complete. The motor fifth and seventh nerve supplies were not affected. The left pupil was dilated, the right moderately contracted. The light reflex was obtained on the right side but not on the left side. The left internal rectus and superior rectus muscles were completely paralysed; the left inferior rectus and inferior oblique muscles were weak. Looking far to the left caused lateral nystagmoid movement.

The upper extremities were not affected as regards motion, sensation, and the reflexes. The extensors of the leg were normal and much stronger than the flexors of the leg. The flexors and extensors of the thigh and the sartorius muscle on each side were strong. In the leg the anterior tibial muscle on each side was the only muscle contracting in voluntary action, and the left was weaker than the right. The patellar reflex on each side was exaggerated, the Achilles reflexes and plantar reflexes were lost. Both lower extremities, especially below the knees, were atrophied. Sensation was normal in the right lower limb, in the right buttock, and in the perineum. The man had control of bladder and rectum. Squeezing the testicles seemed to produce pain.

Dec. 16, 1905.—An examination was made on this date by Dr William J. M'Connell, and showed that sensation was not affected in any form in the right lower limb, but tactile sensation was lost in the left lower extremity over the dorsum of the foot and plantar surface as far as the second toe. The anæsthesia extended on the outer surface of the left leg half way to the knee. An area of anæsthesia was found on the posterior surface of the thigh from about four inches below to one inch above the gluteal femoral fold, and from two inches from the perineum almost to the great trochanter. This was the first time any anæsthesia was found in the region of the buttock. In all these areas of anæsthesia sensations of temperature and pain also were lost.

An examination of the eyes made on Jan. 1, 1906, gave the following results :—O.D.V., $\frac{5}{8}$; O.S.V., $\frac{5}{8}$. O.D. pupil, 3 mm.; O.S. pupil, 6 mm.

O.D. reaction free to light and in convergence and accommodation. O.S. No reaction in any way; paresis of all ocular muscles excepting the external rectus; slight impairment of levator palpebræ.

O.D. media clear, fundus negative. O.S. media clear, fundus negative.

March 11, 1906.—Notes were made by me at the time the patient was in my service. He was shown twice in lectures by me chiefly because of bilateral peroneal palsy. He was weak in both lower limbs, but the weakness was much greater in the peroneal distribution on each side. He had a steppage gait, and when sitting with his feet firmly on the floor could not raise the toes well when the heels were on the ground. The case was striking because of the peroneal palsy occurring with cerebro-spinal syphilis.

Condition, March 11, 1906.—He is able to pull up his right lower limb on command, but it is impossible to get him to pull up the left lower limb. Although his stupor is very great he pulls up the right lower limb and probably therefore has weakness of the left lower limb. When the left lower limb is pricked with a pin he flexes it somewhat at hip and knee, the limb therefore is probably weak but not paralysed, and the test of movement is interfered with by the stupor. He has marked bilateral foot-drop, and the lower limbs below the knees are much wasted both in the muscles in the front and back of the legs. The soles of the feet are also wasted. The lower limbs are abnormally flaccid, especially the left. When either lower limb is stuck with a pin the patient gives distinct evidence of discomfort and puts his hand at the place stuck. The patellar reflex is present, but not very prompt on either side, slightly more so on the left. The Achilles jerk is lost on each side. Babinski reflex is absent on each side, the toes not moving in either direction. The cremasteric reflex is not obtained on either side. The muscles of the calves and the peroneal nerves are not tender to pressure. He cannot be tested for tactile sensation because of his mental condition.

Upper limbs: He can raise the right upper limb on command, but cannot raise the left upper limb. He is therefore hemiparetic. The biceps tendon reflex and triceps tendon reflex are prompt on each side, more so on the left. He feels pin

prick when stuck in either upper limb, but he can move the left very feebly. The upper limbs are not wasted. The left side of the face is paralysed and he does not close the left eyelids as well as the right. It is impossible to get him to put his tongue out or to test movements of his eyes. There seems to be weakness of the right external rectus, but this is not positive.

March 17, 1906.—The man was stuporous, and breathing was difficult. Bubbling râles were heard over the chest. The heart was rapid and weak. He died on this date.

The necropsy revealed: pulmonary hypostasis and œdema; chronic adhesive pleurisy, cyanotic induration of spleen, follicular enteritis, hepatic congestion, cyanotic kidney, purulent meningitis.

The results of my microscopical examination of the nervous tissue are as follows:—

While sections from the third lumbar region show the cells of the anterior horns to be normal, with the exception of an occasional diseased cell, those from the lowest lumbar and sacral regions show these cells intensely degenerated. The nuclei are displaced to the periphery; some of the cells contain several vacuoles; chromatolysis is intense; the dendritic processes of many of the cells have disappeared, and the cell bodies are swollen. The round cell infiltration of the pia, although intense, is not any greater at this region than elsewhere in the cord. Both posterior columns are degenerated in the lower lumbar and upper sacral regions, but the degeneration is much greater on the left side, and is of long standing, although recent degeneration in both posterior columns, especially the left, is also present, as shown by the Marchi method.

Sections from the mid-thoracic and lower cervical regions show intense round cell infiltration of the pia, and thickening of the pial vessels, and degeneration of the columns of Goll, much greater on the left side, and slight degeneration of each crossed pyramidal tract. Perivascular round cell infiltration is also found within the cord.

The round cell infiltration of the pia and the thickening of the arteries is very intense over the medulla oblongata, cerebral peduncles, chiasm, and optic nerves. The optic nerves are partially degenerated.

The left third nerve is intensely degenerated, and a small

vessel accompanying the nerve is almost occluded by proliferation of the intima.

The left seventh nerve and sensory part of the left fifth root are also much degenerated; the motor portion of the left fifth root is only partially degenerated. The right seventh and third nerves and the root of the right fifth nerve are slightly degenerated. The contrast afforded by the condition of the two third nerves is very striking.

Right and left peroneal nerves.—Muscle attached to these nerves shows very intense atrophy; the muscle fibres are small, and the connective tissue is increased in amount. The Weigert hæmatoxylin stain shows considerable degeneration of the nerves.

Right and left plantar nerves.—These are partially degenerated. The muscles on these nerves are also much atrophied, and their connective tissue is much increased.

A gumma was found in the right island of Reil.

SUMMARY.

A male, syphilitic, complained of pain in the lower part of the back on the left side, about April 1905. This was followed soon by numbness and pain in the left lower limb, especially in the calf. He entered the hospital July 27, 1905. At that time he presented foot-drop on the left side. No tenderness was felt over the nerve trunks. The voluntary movement was good everywhere except the dorsal flexion of the foot. It is uncertain from the notes whether dorsal flexion of the right foot was affected at this time. Irritation of the sole of the right foot caused flexion of the toes; irritation of the sole of the left foot produced no movement of the toes. The patellar reflexes were a little prompter than normal. Achilles reflexes were absent. The bladder and rectum functionated normally. The pupils were unequal, and light reaction was lost. Objective sensation was normal.

On August 12, 1905, the tibialis anticus muscles alone contracted on attempt at dorsal flexion of the feet, the right also being affected. Tactile anæsthesia was present on the outer parts of the feet and legs, in the area of the first and second sacral roots, more pronounced on the left side. Pain encircled the lower part of the trunk.

On October 22nd the extensors of the legs were normal, the flexors of the legs were paretic. The flexors and extensors of the thighs were normal. The plantar reflexes were lost. Signs of cerebral syphilis and later left hemiparesis developed. The legs below the knees were much wasted. The lower limbs were flaccid, the patellar reflexes later became diminished.

The remarkable features of this case were the bilateral peroneal palsy affecting the left side before the right with the escape of the tibialis anticus muscles, weakness of the flexors of the legs and extensors of the foot, disturbance of objective sensation in the distribution of the first and second sacral roots or peroneal supply, loss of Achilles reflexes, later loss of plantar reflexes and preservation of patellar reflexes and of the function of the bladder and rectum, in a man clearly affected with syphilis of the nervous system, as shown by the history and cerebral manifestations and pathological findings.

A bilateral peroneal palsy, such as this man presented, is most commonly caused by neuritis, and especially neuritis from alcoholism. The absence of tenderness to pressure over the peroneal nerves and their muscle-supply does not exclude the diagnosis of multiple neuritis, as a purely motor neuritis may occur. The escape of the bladder and rectum, also, is in favour of neuritis, but is a feature also of a lesion of the epiconus, especially in connection with the escape of the tibialis anticus muscles. These muscles probably have centres in the spinal cord above those of the other muscles in the peroneal distribution, and may escape in lesions of the spinal cord, as seen frequently in anterior poliomyelitis. They may escape also in lead palsy when the lower limbs are affected, just as the supinator longus muscles often escape when the posterior interosseous distribution is affected from lead, causing wrist-drop; but it is still undetermined whether lead palsy is primarily due to disease of the nerve cells or of the peripheral nerves. Weakness confined to nerve distribution is one of the most diagnostic features of neuritis, but peroneal palsy may be caused by a lesion of the spinal cord in the epiconus. The patient complained of pain in at least the left lower limb, but pain in the limbs is common in syphilitic meningo-myelitis, and probably results from irritation of the posterior roots; it by no means indicates necessarily peripheral neuritis. A diagnosis in this case between multiple

neuritis and a lesion of the epiconus or of the roots pertaining to this region of the spinal cord was difficult.

A lesion of the grey matter of the fifth lumbar, first and second sacral segments of the spinal cord, a region to which Minor has given the name epiconus, gives a very definite clinical picture characterised by the presence of certain symptoms, as well as by the absence of others belonging to lesions of the conus; the conus to be regarded as beginning with the third sacral segment and extending to the end of the cord. There is paralysis of motion and of sensation in the innervation of the sacral plexus, especially in that of the peroneal nerves. These muscles are most atrophied, and electrical reactions in these are most affected. The gait is of the steppage type because of foot-drop. When the lesion extends higher than the first and second sacral segments, into the fifth lumbar segment, the flexors on the back of the thighs and the gluteal muscles are weak, because of the implication of the fifth lumbar segment. The Achilles tendon reflexes and the plantar reflexes are lost. The negative signs are as important in the diagnosis as the positive; the sphincters of bladder and rectum and the sexual functions are not affected, because the conus in which the centres for these muscles and functions are situated, and the white columns above the conus, are not implicated. The patellar reflexes are preserved, and may be exaggerated, because the lesion does not extend into the fourth lumbar segment; the saddle-shaped anæsthesia over the buttocks is not present because of the integrity of the conus. Minor had no cases with necropsy when he wrote his first paper on this subject,¹ nor does he refer to any necropsy in the few cases of epiconus lesions he quotes from the literature. These as well as his own were all traumatic cases. Sensation may be affected in the feet and outer part of the legs about half way to the knees, and possibly also in a narrow strip extending up the back part of the thighs. In his second paper, published in June 1906, Minor reports two cases of poliomyelitis of the epiconus confined to one side, also a traumatic case, but all without necropsy.² A necropsy was not obtained in Bernhardt's case, and this was not a traumatic case.³ The lesion was supposed to be hæmorrhage or

¹ Minor, *Deutsche Zeitschrift für Nervenheilkunde*, vol. xix. p. 331.

² Minor, *Deutsche Zeitschrift für Nervenheilkunde*, vol. xxx. p. 395.

³ Bernhardt, Salkowsky's *Festschrift*, cited by Minor.

myelitis of the epiconus. Cestan and Babonneix's case 4 in their paper is regarded by Minor as one of epiconus lesion, caused by hæmatomyelia.¹ It is not stated by Minor whether or not a necropsy was obtained. A case of Laignel-Lavastine is not regarded by Minor as entirely typical.

A traumatic case of lesion of the epiconus was under my observation a long time, and was reported by Weisenburg. That also was without necropsy.

These cases referred to by Minor seem to be the only instances in literature of lesions of the epiconus, and by far the majority of these cases are the result of trauma. Unless Cestan and Babonneix's case was with necropsy all were merely clinical cases.

In a diagnosis between lesions of the epiconus and the roots pertaining to it, or lesion confined to these roots, the following points are recognised: In lesions of the epiconus the deformity of the vertebræ, if one exists, is at the first lumbar vertebra; the symptoms develop rapidly and rapidly extend, anæsthesia is pronounced, and the sensory disturbances are of the dissociated type; signs of sensory irritation are absent, and the disturbances are bilateral and symmetrical. In lesions of the cauda equina in the roots pertaining to the epiconus, the deformity of the vertebræ, if one exists, is lower; the symptoms begin more slowly and extend more slowly, pain is severe and lasts a long time, and precedes other symptoms, and the disturbances are asymmetrical. Tenderness to pressure is common in the peripheral lesions, but inasmuch as hypersensitiveness is common in meningitis, probably from irritation of the posterior roots, it does not imply neuritis of the peripheral branches.

In my case, the report of which has just been given, the left leg was affected first, but the right was soon implicated; there was no deformity, as there was no trauma; the symptoms developed rapidly, soon reached their height, and remained stationary some time without involving either upper limb until cerebral hemiparesis occurred. Anæsthesia was pronounced, but dissociation of sensation was not present, and the implication was bilateral and symmetrical. The symptoms were therefore suggestive of a cord lesion. Even with the microscopical study before us it is difficult to say whether the multiple neuritis

¹ Cestan and Babonneux, Case 4, cited by Minor.

occurred first and the cellular changes in the lower lumbar and sacral regions were secondary, in the form of a reaction at distance, or whether the roots of the peroneal nerves arising in the epiconus were first affected, as they may have been, by the meningomyelitis. No greater intensity of the meningitis is present in the lower lumbar and sacral regions to explain the implication of the roots of these regions and the escape of roots from higher levels. It is possibly more reasonable, therefore, to assume that the peroneal nerves were the first affected, and that the case was one of syphilitic multiple neuritis occurring with syphilitic meningo-myeloencephalitis. With this explanation we can understand why the nerve-cells of the anterior horns of the upper lumbar region afforded such a striking contrast to those of the lower lumbar and sacral regions.

Two other cases of the epiconus symptom-complex have come under my observation :—

CASE II.—G. Iver., aged 35 years, a patient of Dr Stengel, was admitted to the University Hospital, April 27, 1907. In the middle of February 1907, he was taken ill with a high fever. When seen by a physician a few days later he had an enlarged spleen and rose-spots. The temperature went down to normal, and he was able to work at the end of a week. He worked about ten days, when he again had fever, and the symptoms indicated incipient typhoid fever, although the spleen was not enlarged and rose spots were absent. He rapidly grew worse, and developed meningeal symptoms, with positive Koenig's sign and ankle clonus. He was stuporous for two weeks. This condition disappeared, and was followed rapidly by pneumonia of the lower left lung, which resolved very slowly. The pneumonia occurred about March 20th. Symptoms of empyema on the same side followed the pneumonia. About April 6th pus was obtained by needle, and soon after this he coughed up large quantities of muco-purulent material, and this he continued to do.

May 12, 1907.—Examination by Dr Spiller.

The lower limbs have good voluntary power except in the peroneal distribution on each side. Bilateral foot-drop is present, slight on the right side, but very pronounced on the

left side. He is able to dorsally flex the right foot, even to a moderate degree of resistance, but in attempting to dorsally flex the left foot contraction occurs only in the anterior tibial muscle. The muscles of the legs below the knees are wasted. He has no fibrillary tremors. The patellar tendon reflex is exaggerated on each side, and patellar clonus is present on each side, ankle clonus also, but the latter is soon exhausted. Ankle clonus with pronounced foot-drop on the left side is very striking. Sensations of touch and pain are normal in the lower limbs. Babinski's sign is not obtained on either side in a characteristic manner, but on the right side at times all the toes except the big toe are extended. Babinski's reflex is not indicated on the left side by extension of any of the toes. Cremasteric reflex is weak on the left side, prompt on the right side. Sensations of touch and pain are normal about the anus and in the perineum. He has no pain nor tenderness in the lower limbs. Micturition and defecation are normal.

The grasp of the hands is good. The biceps tendon reflex and triceps tendon reflex are exaggerated on each side. Sensations of pain and touch are normal in the upper limbs. No wasting of hands or forearms. Voluntary power in the upper limbs is good.

He closes the eyelids, shows the teeth, and draws up the corners of the mouth very well. Pupils are equal, and respond promptly to light and in convergence. Extraocular muscles are normal. The tongue is normal. Speech is that of a patient weak from sickness, not from organic nervous disease.

Diagnosis.—Lesion of epiconus, poliomyelitic in character, following pneumonia.

CASE III.—F. S., aged 42 years, male, was injured November 6, 1905, by falling and striking his back in the lumbar region. At the present time, October 1907, sensations of pain and temperature are diminished, but not lost, over the outside of each leg below the knee, and on the dorsum and sole of each foot, especially on the right side, and are normal on the inner side of each leg and back and front of each thigh. Tactile sensation is normal in the lower limbs. The patellar reflex is present on each side, but much diminished, and is shown only by contraction of the quadriceps muscles. The Achilles tendon

reflex is nearly normal on the right side, but is very weak on the left side. Complete foot-drop is present on each side. Babinski's sign is not present on either side. The flexors on the back of the thighs are a little weak. The functions of bladder and rectum, and of the sexual organs, are not impaired. Sensation about the anus and down the back of each thigh is intact.

I call attention to the preservation or even exaggeration of the Achilles tendon reflexes in certain cases presenting the epiconus symptom-complex. It may indicate that the centres for these reflexes are at a higher level. Exaggeration of tendon reflexes from a lesion below the reflex arcs I have seen repeatedly.

Abstracts

ANATOMY.

THE RETICULAR APPARATUS OF GOLGI-HOLMGREN
 (54) **STAINED BY NITRATE OF SILVER.** (L'appareil réticulaire de Golgi-Holmgren coloré par le nitrate d'argent.) S. R. CAJAL, *Trav. du Labor. de Recherches Biol.*, T. v., F. 3, 1907, p. 151.

CAJAL has recently, by his new silver method after previous fixation with formalin, succeeded in staining the Golgi-Holmgren reticulum in the cells of the ganglia of the spinal cord of the newly-born dog or cat. The reticulum appears as a series of intercommunicating tubes with dilated portions alternating with segments so thin as to be almost imperceptible. The network is found fairly uniform throughout the whole of the cell-body, and for a short distance into the dendrites. The structure of the reticulum varies with different kinds of cells, being specially extensive and complicated in the large motor cells. The fibres, especially in the dilated parts of the reticulum, show a pale, almost homogeneous, slightly stained part in the interior, with a marginal layer more deeply stained and of a granular appearance.

Cajal interprets this as a channeled system filled with some coagulated substance which has a faint attraction for colloid silver. He regards the apparatus described by Golgi and that of Holmgren as being identical.

The reticulum is found in every kind of nerve cell, and even in the ependymal epithelium. The differences in staining seem to show that the chemical properties of the reticulum are not absolutely identical in all vertebrata.

ALEXANDER BRUCE.

ON A NEW ORIGIN OF THE PEDUNCULAR BUNDLE OF

(55) **TÜRCK.** (*Sopra una nuova origine del fascio peduncolare del Türk.*) PUSATERI, *Riv. Ital. di Neuropatol., Psichiat. ed Elettroter.*, Vol. i., F. 1, p. 29.

IN the examination of a case of softening in the median, and to some extent in the anterior part of the second and third temporal convolutions, Pusateri found a degeneration of the bundle of Türk. He argues, not very convincingly, that this case shows a wider origin for the bundle than that given by Dejerine, viz., the median part of the second and third temporal convolution.

ALEXANDER BRUCE.

A NEW SELECTIVE STAIN FOR THE NERVOUS SYSTEM.

(56) (*Eine neue elektive Nervensystemfärbung.*) RENÉ SAND, *Arb. a. d. Neurol. Instit. a. d. Wien. Univ.*, Bd. 15, Teil 1, 1907, p. 339.

FOR this modification of the silver method the author claims certainty and constancy of results with both normal and pathological material. The staining is not done *en bloc*, but on paraffin sections; and further, the method of fixation is such that sections from the same block can be stained by Nissl's method, and by selective stains for leucocytes, connective tissue or neuroglia, or by general stains such as hæmalum, carmine, etc.

The method for impregnating the axis cylinders is as follows:—

Pieces of tissue to be obtained as fresh as possible and not over 5 mm. thick.

Fix in a mixture of pure anhydrous acetone 90 c.c. and concentrated pure nitric acid 10 c.c. for forty-eight hours, the mixture being changed after one, four, and twenty-four hours.

Transfer to pure anhydrous acetone for six to eight hours, changing three times. Then place directly in a paraffin bath at 50° C. for two hours, changing twice.

Sections of 10 μ fixed on slide with albumen, cleared successively with xylol and acetone, and placed directly in 10 per cent. freshly prepared silver nitrate solution for twenty-four hours, in oven at

30°-38° C. Then prepare the following solution:—To 50 c.c. 10 per cent. fresh watery silver nitrate solution add strong ammonia until the ppt. which is formed is just dissolved and no more. The sections, which should be still uncoloured or only faintly yellow, are washed for a second or two in distilled water and put in the ammoniac-silver solution for forty-eight hours, till distinctly grey-brown in colour.

Wash thoroughly in distilled water.

Tone for five to ten minutes till steel-grey in colour in the following bath:—3 c.c. 1 per cent. gold chloride solution, 17 c.c. 2 per cent. ammonium sulphocyanide solution, 80 c.c. aq. distill.

Wash in water.

Flush for fifteen secs. with 5 per cent. sod. hyposulphite solution. Wash very thoroughly in distilled water, pass through alcohol or acetone, mount in balsam in xylol.

The sections should be free from all precipitate; the axis cylinders impregnated grey to black; glia and connective tissue slightly greyish but transparent; elastic fibres and muscle fibres impregnated like the axis cylinders, but not so deeply. Nuclei are faintly stained. Nerve cells are grey, and their fibrillary reticulum is often well brought out. The neurofibrillæ and fine unmedullated fibres are not stained by this method.

The paper also gives shortly the other selective stains which may be applied to sections fixed by this method.

J. H. HARVEY PIRIE.

PHYSIOLOGY.

A CONTRIBUTION TO THE STUDY OF THE RELATION

(57) BETWEEN LABYRINTH AND EYE. (*Beitrag zur Lehre von der Beziehung zwischen Labyrinth und Auge.*) C. BIEHL, *Arch. a. d. Neurol. Instit. a. d. Wien. Univ.*, Bd. 15, Teil 1, 1907.

It is now sufficiently established that various conditions of the labyrinth, as well as stimulation of that organ, have influence on the position and on the movements of the eyes. The question on what special functions of the ocular muscle the influence is exerted remains unanswered.

In the author's own experiments, attention was chiefly directed to a sign which should be known as the H. Hertwig Majendie squint position. This consists of a vertical divergence and of an associated lateral turning of the eyeballs.

The object of the investigation was to study the results of stimulation and division of the vestibular nerve isolated at the base of the brain, with special reference to the effects on the oculomotor

apparatus and by means of the resulting nerve degeneration to trace the course of the vestibular fibres.

The experiments were made on sheep. Eleven sheep were chosen aged about four weeks. They were allowed to live for not more than twenty-eight days after the operation.

The effects of stimulation were remarkably uniform; first, horizontal nystagmus towards the same side was induced, followed rapidly by an associated change in the position of both eyes, namely, a lateral turning to the same side and also a vertical divergence, the eye on the operated side being raised and on the other lowered. After division of the nerve the nystagmus continued or almost disappeared, but the eyes took up an exactly opposite position—a typical H. Hertwig Majendie squint—namely, lateral turning to the opposite side and vertical divergence with lowering of the eye on the side of the operation and raising of the other eye. This sometimes reached a very marked degree. These appearances rapidly passed off and disappeared entirely within a week.

The results of the microscopical investigation were similar to those obtained by Ramon y Cajal and Held.

The rest of the paper is devoted to a discussion of the physiological significance of the innervation of the labyrinth to vertical divergence.

W. G. PORTER.

SOME OBSERVATIONS ON THE BEHAVIOUR OF THE AUTO-

(58) MATIC RESPIRATORY AND CARDIAC MECHANISMS AFTER COMPLETE AND PARTIAL ISOLATION FROM EXTRINSIC NERVE IMPULSES. B. G. N. STEWART, *Amer.*

Journ. Physiol., Vol. xx., 1907, p. 407.

BOTH vagi were divided in dogs, cats, and rabbits simultaneously or at different times, and the effects on the cardiac and respiratory rhythms observed. The ratio of pulse rate to respiratory rate, which is at first much increased through quickening of the heart and slowing of the respiration, tends to diminish as time goes on, the change being due to a fall in the pulse rate, the rate of respiration in the great majority of cases showing no tendency to increase.

The author believes that when all its afferent channels are cut off, the respiratory centre discharges impulses automatically at the rate of about four per minute. This has been studied mainly in the cat, and it appears to be remarkably constant not only in different individuals of the same species, but also in the different mammalian species investigated.

The existence of accelerator fibres in the vagus of the dog was clearly demonstrated in many of the experiments.

SUTHERLAND SIMPSON.

ON THE AFFINITY OF THE SPINAL CORD FOR STRYCHNINE

(59) **AND COCAINE.** (Ueber die Entgiftung von Strychnin und Kokain durch das Rückenmark.) B. T. SANO, *Arch. f. d. gesam. Physiol.*, Bd. 120, H. 6, 7, 8, 9, 1907, S. 367.

THE power of the different parts of the spinal cord to combine with strychnine and cocaine has been investigated by the author. The white matter possesses this property in a higher degree than the grey matter. The anterior part of the grey substance has a greater affinity for strychnine than the posterior part, and vice versa for cocaine. The behaviour of the different regions of the cord in their affinities for these substances has no relationship to the blood supply. The general morphological and functional characters of the cell elements of the grey matter have a chemical basis, and this chemical differentiation probably also extends to the nerve fibres taking origin in these cells.

SUTHERLAND SIMPSON.

ACTION OF ACONITINE ON NERVE-FIBRES. By A. D. WALLER

(60) (*Proc. Physiol. Soc.*), *Journ. Physiol.*, Nov. 29, 1907, p. xxx.

THE author finds that this substance has an action on nerve fibres as well as on nerve endings. Even 1 in 100,000 injected subcutaneously in a frog is sufficient to abolish the negative variation in the nerve fibres.

SUTHERLAND SIMPSON.

THE RELATION OF AFFERENT IMPULSES TO FATIGUE OF

(61) **THE VASOMOTOR CENTRE.** By W. T. PORTER, H. K. MARKS, and J. B. SWIFT, *Amer. Journ. Physiol.*, Vol. xx., 1907, p. 444.

THE authors attempted to fatigue the vasomotor centre, in anæsthetised or decerebrate cats, dogs, and rabbits, by the stimulation of afferent nerves for prolonged periods, but were not successful. At the end of a three hours application of the interrupted current to the posterior root of one of the lumbar nerves, *e.g.*, there was only a slight fall in the general blood pressure—no more than in a control animal (cat) subjected to the same manipulations except stimulation of the nerves.

SUTHERLAND SIMPSON.

PATHOLOGY.**DEGENERATION AND REGENERATION OF THE END-PLATES**

- (62) **OF MUSCLE AFTER SECTION OF THE NERVES.** (Dégénération et Régénération des Plaques Motrices après la Section des Nerfs.) TELLO, *Trav. du Labor. de Recherches Biol.*, T. v., F. 3, 1907, p. 117.

TELLO has studied this question principally in the rabbit, the lizard, and the frog, by means of the silver method after previous fixation with ammoniated alcohol, using pyrogallie acid and formalin as the reducing agents. The chief conclusions from his work, which is not yet finished, are :

(1) That the peripheral part of a divided motor nerve and its termination in the muscle begin to degenerate in twelve to fourteen hours after section, and continue doing so until all the remains of the degenerated fibres are absorbed. Absorption in the end-plates is completed in two and a half days, and in the nerve fibres in from two or three days to a month or more.

(2) The phases of the degeneration of the nerves and end-plates are hypertrophy of the terminal arborisations and the neuro-fibrillæ, excessive staining of the interfibrillary plasmatic substance with granular appearance of the argentophile substance, decomposition of this into granules, and ultimately fragmentation of the branches.

(3) All the detritus of the plaques is removed, perhaps by digestion of the granular substance and nuclei ; that in the end-plates by the cells of Schwann.

(4) Certain fibres, which may survive forty-eight hours, show in the end-plates a return to the embryonic condition, and consequently, instead of disintegrated branches, show a terminal sphere, which is destroyed later.

(5) In cold-blooded animals the degeneration is more delayed.

(6) The regenerating fibres which start from the central end commence to reach the muscle almost two and a half months after the operation in rabbits of two to three months old, and a month or a month and a half after section in new-born rabbits.

(7) The regenerated fibres enter the old nerve tubes, probably attracted by the positive chemotactic substance produced by the cells of Schwann. These fibres always terminate in clubs.

(8) The young fibres divide at several different parts of their course and form a large number of daughter-fibres, which sometimes separate and continue their course in different tubes, but they frequently follow a long course within the same sheath. Ultimately they cease to be invested by a sheath, and come into contact with

muscular fibre, in which they terminate by a bud, in the substance of which the arborisation of the end-plate is produced.

(9) Each new fibre gives origin to a large number of plates, either collateral or terminal, which confirms the fact that only a small number of nerve tubes reach the muscle, and that this small number is supposed to innervate the whole of the muscle fibres.

(10) The nuclei and the already existing plates are conserved with unimportant variations, and they attract by chemiotaxis one of the newly-arrived fibres of the motor nerve. When the terminal bud has reached the granular substance, it divides into an increasingly complicated ramification.

(11) Some fibres, before coming into contact with the corresponding plates, turn backwards within their guiding sheaths, sometimes within the same sheath of Henle, sometimes following a more complicated course. This uncertainty of path of the fibres is explained by the abundance and complexity of the chemiotactic currents.

ALEXANDER BRUCE.

ON PATCHY ATROPHY OF THE MEDULLARY SHEATHS IN

(63) **THE CORTEX OF GENERAL PARALYTICS.** (Ueber den fleckweisen Markfaserschwund in der Hirnrinde bei progressiver Paralyse.) O. FISCHER (of Prague), *Arch. a. d. deutschen psych. Univ.-Klinik in Prag*, 1908.

TUCZEK and subsequent observers demonstrated in the cortex of general paralytics a fairly diffuse affection of the medullary sheaths, which is especially well marked in the outer layer. In 1899 Siemerling called attention to the presence in three cases of general paralysis of perivascular foci of absence of the medullary sheaths; similar communications followed. Fischer in this article gives the results of his studies of these patches of disappearance of the medullary sheaths in great detail; his material consisted of forty-three cases of general paralysis, and of fifty-seven other brains; the method of serial sections was employed. In 65 per cent. of the cases of general paralysis these patches were present; in 35 per cent. none were found. In none of the cases except those of general paralysis were these patches observed. Histologically they are quite different from the light patches seen in senile brains. They cause no secondary degeneration, as the axis cylinder is not affected. The patches are therefore similar to the foci in disseminated sclerosis, but present no inflammatory reaction and much less glia reaction than the latter. This atrophic condition of the medullary sheaths shows no relation to any special inflammatory reaction; the author uses this to support the view that the parenchymatous degeneration and the inflamma-

tory element in the picture of general paralysis are two independent processes. The atrophy of the medullary sheaths in the special patches is similar in nature to the diffuse medullary atrophy in the cortex and represents therefore merely a special localisation of the morbid agent. There is no question of the foci being artefacts, as they are in certain cases visible in the fresh unfixed brain.

The article is illustrated by eighteen excellent figures.

C. MACFIE CAMPBELL.

A STUDY OF THE NEUROFIBRILS IN DEMENTIA PARALYTICA, DEMENTIA SENILIS, CHRONIC ALCOHOLISM, CEREBRAL LUES, AND MICROCEPHALIC IDIOCY. S. C. FULLER (of Westborough, Mass.), *Amer. Journ. of Insan.*, April 1907.

A CAREFUL study has been made of the cerebral cortex in fifty-four patients dying insane at the Westborough Insane Hospital. The paper is illustrated with thirty excellent plates. The author emphasises the extreme importance of caution in the interpretation of the alterations in neurofibrils in material from pathological sources. He believes, however, that pathological changes may be demonstrated in the neurofibrils of the cerebral cortex in all persons dying insane.

His conclusions are:—(1) The alterations in the neurofibrils taken alone have no more value for diagnostic purposes than the mere disintegration of the tigroid masses in a Nissl preparation. (2) Poverty in cell processes, the more or less universal tinging of the nucleus, and destruction of the finer intercellular fibrils, characterise the silver impregnation of the dementia paralytica cortex; fair preservation of the dendrites and an equally diffuse destruction of intercellular fibrils, but without special preference for the finest elements, is the rule in dementia senilis. (3) The alterations in the remaining groups of cases reported suggest that these changes may be due to a variety of causes, such as oedema, faulty nutrition or development, and the direct action of intoxications introduced from without. (4) Alterations in the neurofibrils, such as granular disintegration, fragmentation, localised swellings, rarefaction, and complete destruction were to be found in varying stages of intensity in all of his cases. (5) Just as the ensemble in a Nissl preparation is of value in determining dementia paralytica, or in distinguishing a luetic meningeal or perivascular infiltration, in almost to the same degree may the sum of the findings in a silver preparation for neurofibrils be employed in making an anatomical diagnosis of dementia paralytica, or in differentiating it from a disease with a dystrophic substratum, such as senile dementia.

C. H. HOLMES.

CLINICAL NEUROLOGY.

ALCOHOLIC NEURITIS. (*Névrite alcoolique.*) BABINSKI (of (65) Paris), *Jour. des Prat.*, No. 47, Nov. 23, 1907.

A MAN, fifty-four years of age, waiter in a café, presented a series of mental, sensory, and motor symptoms, which might possibly have been taken as indicating the presence of tabes, complicated by general paralysis.

Careful examination showed the incorrectness of such a diagnosis, for while the patient suffered from loss of memory and intellectual weakness, with mental confusion, the disturbances of speech peculiar to general paralysis were absent, and the wavering memory was subject to complete returns. Further, the recollections of recent date were alone lost, whereas these persist in general paralysis. The psychic troubles were simply of alcoholic origin.

The patient had had syphilis: but in considering the question of the presence of tabes one noted that such pains as the patient suffered from were different from the lightning pains of that disease. Further, the muscles of the calf were tender when pressed, and the passage of an electric current through them caused sharp pains—not as obtains in tabes, where neither pressure nor the electric current cause pain. The disturbances of tactile and deep sensibility, frequent in tabes, were here absent. Also the muscular force was affected. In walking the foot was raised with difficulty, and allowed to fall in stepping on account of the muscular atrophy on the anterior aspect of the limb. The pupils and sphincters were unaffected.

The symptoms were purely toxic in origin, alcoholism producing a peripheral neuritis with mental confusion. The diagnosis in such cases is of course of great importance from the point of view of prognosis.

A. HILL BUCHAN.

ANTERIOR POLIOMYELITIS IN THE ADULT, WITH ILLUSTRATIVE CASES. G. L. WALTON, *Boston Med. and Surg. Journ.*, Nov. 28, 1907, p. 719.

IN this paper short notes are given of ten typical cases of acute or sub-acute anterior poliomyelitis in young adults. The author emphasizes the fact that the disease is essentially the same as in children, although the onset of paralysis is usually more retarded, and the extension of paralysis from one group of muscles to another is less immediate. The real seat of infection is apt to be obscured by temporary symptoms pointing to the meninges; the implication

of which, he thinks, is probably independent, not an extension from the cord substance. The prognosis for recovery seems better than in children ; in four of these cases it was practically complete.

J. H. HARVEY PIRIE.

ACUTE POLIOMYELITIS AND ALLIED DISEASES. (Akute (67) Poliomyelitis und verwandte Krankheiten.) HARBITZ and SCHEEL, *Deut. med. Woch.*, Nov. 28, 1907, p. 1992.

THE authors of this paper have made a full anatomico-pathological study of seventeen cases occurring in Norway, during the epidemic of 1903-6. Thirteen of these cases died during the acute stage of the illness, four subsequently. Ten cases were children, the remainder young adults. They did not confine their investigation to the spinal cord, but made a comprehensive survey of the whole nervous system. They conclude that in fatal cases inflammatory poliomyelitis is found to extend over the whole cord, medulla and pons as well as the greater part of the brain. Probably the same holds good for non-fatal cases. There is always also some meningitis, and indeed this is primary, the inflammation extending inwards from the pia along the sheaths of the vessels. When the inflammation is marked in various parts, then we get clinical types which are no longer classified under acute poliomyelitis, but are really the same disease. Transitional types may be seen between poliomyelitis and bulbar paralysis, meningo-encephalitis and transverse myelitis. No transition to polyneuritis or epidemic cerebro-spinal meningitis has been observed.

The microscopic appearances described, beyond the extent over which they occur, are familiar, and need not be detailed. No organisms were found.

J. H. HARVEY PIRIE.

AMYOTROPHIC LATERAL SCLEROSIS, WITH HEMIPLEGIC (68) ONSET, IN A BOY OF SIXTEEN. (Sclérose latérale amyotrophique, à début hémiplégique chez un sujet de 16 ans.) BOUCHAUD, *Journ. de Neurol.*, déc. 5, 1907, p. 465.

AUTHOR describes a case in a boy of sixteen, in which there was a somewhat rapid development of atrophy on the left side, especially in the upper limb, associated with an exaggeration of tendon reflexes on the same side. As the condition is associated with some sensory phenomena, it is doubtful if it can be regarded as a pure case.

ALEXANDER BRUCE.

PRURITUS IN TABES. (*Pruritus bei Tabes.*) GÜNZBURGER,
(69) *Münch. Med. Wchn.*, Dec. 31, 1907, p. 2642.

THE recent discussion on this subject at the Soc. méd. des Hôp. (v. *Rev. of Neurol.*, Dec. 1907, p. 909) reminds Günzburger of a case that occurred in his own practice two years ago. His patient suffered, especially at night, from severe pruritus, which was most marked in the lumbar region. Treatment for scabies afforded no relief. There were no other subjective disturbances. Examination revealed complete absence of the knee-jerks, the presence of Romberg's sign and reflex iridoplegia. The patient, in fact, had suffered from tabes for years without knowing it. Günzburger concludes that in every case of pruritus the reflexes as well as the urine should be tested.

J. D. ROLLESTON.

CASE CONTRIBUTION ON SKULL INJURIES. (*Beitrag zur*
(70) *Kasuistik der Schädelverletzungen - Basisfraktur, Contusio cerebri, traumatische Epilepsie und Demenz; aphasische Symptome.*) G. ACKERMANN, *Monatsschr. f. Psychiat. u. Neur.*, Bd. 22, Ergänzungsheft, S. 1.

THIS is a full description of a case with a few lines of comment thereon. The main phenomena are sufficiently indicated in the title; a transitory right hemiplegia also occurred. The accident took place during the convalescence from a septicæmia.

ERNEST JONES.

STUDIES IN MENINGOCOCCUS INFECTIONS. D. DAVIS, *Journ.*
(71) *Infect. Dis.*, Nov. 15, 1907.

DAVIS has made a very thorough study of eleven cases of cerebro-spinal meningitis, which were apparently sporadic in nature. He was fortunate enough to find the meningococcus in the blood of two out of nine cases examined. The two positive cases were in the first four days of their illness, and Davis thinks it probable that systematic cultivation of large quantities of blood at this early period of the disease would probably frequently give positive results. In one of these cases the meningococcus was isolated from the nasal mucus, the discharge from the eyes, and the cerebro-spinal fluid as well as from the blood, yet in spite of this very marked and general infection the patient made a good recovery.

As regards the path of infection Davis is inclined to favour the theory that the infection is taken originally into the nose and mouth. This view does not necessitate the adoption of the opinion that thence the meningococcus penetrates to the meninges through the

base of the skull. We only have to assume it enters the lymph or blood stream and localises itself in the cord and brain, just as the cholera organism selects the intestine. Davis does not think the infection is gastro-intestinal in origin, believing the micro-organism too sensitive to acids to make such a path of infection probable. In favour of the other route he instances cases in which the meningococcus was found in the blood before any cerebro-spinal symptoms occurred. It is possible, then, that localisation of the organism occasionally fails to ensue.

In 200 examinations of the nose and throat of 150 normal persons, Davis failed to find the meningococcus. So, although the carrying of infection by contacts is admitted, how are we to explain sporadic cases occurring at long intervals? Normal people do not apparently harbour the germ when there is no epidemic, and the difficulty is increased when we remember that the meningococcus is short-lived and difficult to cultivate. The only suggestion, and that a purely theoretical one, is that the Gram negative diplococci, which superficially resemble the meningococcus, but which culturally are distinguishable from it, and which are found in normal noses and throats, may under certain circumstances become modified and acquire the characteristics of the meningococcus.

Davis also made interesting experiments on the agglutinins of the disease. Eight cases were examined. All gave positive reactions with a 1-50 dilution sooner or later in the course of their illness. The highest dilution with which a positive reaction was obtained was 1-500, and this was at the end of the fourth week of the disease. The phenomenon is not to be expected till the end of the first week, when it may occur in 1-25 dilution. The agglutinins are thermostable, resisting 65° C. for one hour. They are not contained to an appreciable amount in the cerebro-spinal fluid.

Defibrinated meningitis blood rapidly killed meningococci introduced into it, plates being usually sterile in three hours. The number of leucocytes in the blood made no difference to the rapidity of their destruction. Again, with a normal blood, in which the meningococci rapidly multiplied, killing the leucocytes by heat made no difference to the rapidity of their multiplication. Opposed to these destructive processes there are other elements in the blood which favour growth, as, while the meningococcus will not grow in cerebro-spinal fluid, its growth is facilitated if red corpuscles are added to the mixture.

Davis in a previous paper reported that he had found but little opsonic activity in cerebro-spinal meningitis. He now admits that there is more phagocytosis in meningitis blood than in normal blood. Much would appear to depend on the strain of meningococci employed. On the whole the patient in the fourth week of the illness shows a marked increase in opsonic power.

Further experiments were made to note the effect of the injection of the meningococcus into animals and man. Davis, like other observers, found that the organism has very little virulence for animals if injected subcutaneously or intravenously. Intraperitoneal injections, however, killed a high proportion of small animals in about twelve hours. Flexner's plan of intra-lumbar injection does not appear to have been attempted.

Two patients were injected with cultures killed by heat. The first, treated with this vaccine in the sixth week of the illness, received the scrapings of several serum cultures in 3 cc. of salt solution. No subjective symptoms followed. There was, however, some local irritation at the seat of injection. Some days later a second injection was given and was followed by a small sterile abscess. A leucocyte rise occurred after each injection to fall to normal in about three days. The reaction after the second injection was more marked. The temperature fell about two days after this, and remained steadily normal, the patient making an uninterrupted recovery. The second patient received only one injection with no immediate result but a mild local reaction. A rise of temperature and some leucocytosis occurred two days later. Thereafter the patient became worse and died five weeks after injection in the tenth week of the disease.

The most striking of all the results obtained by Davis is the effect of an injection of a similar sterilized culture administered to himself. Immediately there was local irritation and smarting pain. In twenty minutes he was seized with nausea and vomiting. Ten minutes later he had a rigor lasting half an hour. Thereafter headache, muscular pain, purging, and vomiting. In three hours from the injection his temperature was 103° F. The vomiting continued throughout the night, accompanied by marked prostration. Next day there was some improvement. A diffuse rash, but no hæmorrhages, appeared all over the body. Temperature varied from 101° to 102° F. The face was flushed and there was some stupor. On the following day the temperature was a little lower, the rash disappeared. On the fourth day herpes appeared on lips, eyelids, and palate. From this point onwards there was improvement, although it was a month before the evening temperature was steadily normal. The fever was accompanied by a steady rise in the number of leucocytes and in the opsonic index.

Davis concludes, then, that patients suffering from cerebro-spinal meningitis acquire immunity slowly. If a vaccine is to be given early in the illness only small doses should be employed, and these may be gradually increased. Injections, judging from the opsonic and leucocyte curves, may be given at intervals of from six to eight days. Something is to be hoped from treatment on such lines.

CLAUDE B. KER.

CEREBRO-SPINAL MENINGITIS. CANTLEY, *Brit. Journ. Child. Dis.*, Nov. 1907.

CANTLEY gives an interesting analysis of 125 cases of meningitis observed by him. Of these 71 were tubercular, 36 cerebro-spinal, 11 pneumococcal, while 5 were due to ear disease. In reference to the latter he considers that the importance of ear inflammation as a source of meningitis in early life has been much exaggerated. Sometimes the ear trouble found in such cases is merely a part of a general infection and is in no sense causative. On other occasions the ear inflammation may be due to extension from the inflamed meninges. As regards mortality the tubercular cases were the most fatal, and, as we would expect, the cerebro-spinal ones the least so. Ten out of eleven pneumococcal cases succumbed.

Age is of some importance in diagnosis. If meningitis occurs in the first six months of life it is probably not tubercular. In the second six months, not more than one-third of the cases are tubercular. After the first year is over, the vast majority of the cases are tubercular. In cerebro-spinal fever, vomiting and diarrhoea are more common than in the tubercular type and the pulse is more rapid.

Severe sporadic cases of cerebro-spinal meningitis may occur with hæmorrhages and other acute symptoms which render these indistinguishable from the epidemic form. On the other hand, during epidemics cases conforming to the post-basic type of meningitis often occur. There is no dividing line. Cantley, like Osler, compares outbreaks of cerebro-spinal fever to those occasions where pneumonia appears to take on an epidemic form.

CLAUDE B. KER.

HERPES IN CEREBRO-SPINAL MENINGITIS IN CHILDREN.

(73) (*L'erpete nella meningite cerebro-spinale nei bambini.*) GIOSEFFI, *Riv. di Clin. Pediatr.*, Nov. 1907, p. 920.

A BOY, aged eight months, was admitted to hospital on the third day of an attack of cerebro-spinal meningitis. Some vesicles were present at each internal canthus and on the left cheek. Lumbar puncture performed the same day revealed markedly turbid fluid containing almost exclusively polymorphic leucocytes, and many intra- and extra-cellular diplococci which on culture proved to be meningococci. On the following days the child got worse, and the herpetic eruption became much more extensive. Fresh vesicles appeared almost every day, covering the right ala nasi and bridge of the nose, the right cheek, temple, and upper lip. The mucous membranes were also invaded. The vesicles spread along the right side of the tongue to the tip and covered the hard and soft palate.

Both eyelids were also affected. Some vesicles formed on the cornea, and as they desiccated left small opacities. Finally the meningeal symptoms increased, pneumonia developed, and death took place within ten days of the onset.

J. D. ROLLESTON.

THE TREATMENT OF EPIDEMIC CEREBRO-SPINAL MENIN-

(74) **GITIS.** (Zur Therapie der Meningitis cerebro-spinalis epidemica.) TÖBBEN, *Munch. Med. Wchn.*, No. 49, p. 2420.

OF 66 cases of epidemic cerebro-spinal meningitis 31 died, a mortality of 47 per cent. Thirty-seven of these were treated by lumbar puncture only with 21 deaths, a mortality of 56·7 per cent. Lumbar puncture was adopted first for diagnostic purposes, and was afterwards repeated for increase of pyrexia, severe headache, vomiting, or somnolence; 25-50 c.c. were withdrawn. The amelioration produced by lumbar puncture was often striking, though it was difficult to determine whether it had a curative effect on the disease. The remaining 29 cases, which were treated with both lumbar puncture and Kolle-Wassermann's serum, furnished a mortality of 34·5 per cent. (10 deaths). The earlier the serum treatment was adopted, the better the result. Among 12 first or second day cases, there were two deaths (16·6 per cent. mortality). Among four third or fourth day cases, there was one death (25 per cent. mortality). Among the 7 fifth to seventh day cases, there were three deaths (42·9 per cent. mortality). Among the six cases on which treatment was started on the eighth day or later there were four deaths (66·6 per cent. mortality). Serum rashes occurred in 3. Children under 3 received 5 c.c., older children 10 c.c., and adults 30 c.c. The doses were repeated as occasion demanded.

J. D. ROLLESTON.

ON THE DIAGNOSIS OF THE SYPHILOGENOUS DISEASES OF

(75) **THE CENTRAL NERVOUS SYSTEM IN THE EARLY AND ADVANCED STAGES.** (Ueber die Diagnose und Frühdiagnose der syphilogenen Erkrankungen des zentralen Nervensystems.) W. ERB, *Deutsche Zeitsch. f. Nervenheilk.*, 1907, p. 425.

THE intimate causal connection between syphilis and tabes is now almost universally recognised, but there are still numerous problems awaiting solution. Erb here discusses the diagnosis of incipient, incomplete, or abortive forms of tabes.

Firstly, as to the significance of loss of the pupillary reflex to light, whilst Babinski and other French authors regard it as a sign

merely of antecedent syphilis, Moebius regarded it as an indication of incipient tabes. Erb recounts a series of seven cases in which for a long time, sometimes as long as twenty years, the Argyll-Robertson phenomenon was the only pathological sign present, but in which other symptoms of tabes subsequently developed. He also gives particulars of eight cases in which the Argyll-Robertson phenomenon has been present for periods varying up to twelve years, without any other evidence of tabes. In two of these cases the cerebro-spinal fluid was examined and found normal (one of them, however, had absence of all the deep reflexes). Erb concludes that whilst the Argyll-Robertson pupil by itself is highly suspicious of incipient tabes, it is not conclusive, and we must be on the alert for other evidence, such as hypæsthesia and hypalgesia of radicular distribution, excessive sensitiveness to cold on the trunk, paræsthesiæ of the genitals or in the ulnar area, etc., also various "crises," minor degrees of ataxia and of hypotonia, together with the condition not merely of the knee-jerks, but also of the ankle-jerks and of the deep reflexes of the upper limbs.

He agrees as to the immense diagnostic value of examination of the cerebro-spinal fluid, especially its cytological examination. Lymphocytosis is present in upwards of 95 per cent. of cases of tabes. This lymphocytosis he believes to be due to meningeal irritation, syphilitic in origin, whether diffuse or localised. But whilst the occurrence of lymphocytosis is strong evidence of previous syphilitic infection, its absence does not absolutely exclude syphilitic disease of the central nervous system.

The three clinical phenomena of Argyll-Robertson pupil, tabes, and lymphocytosis may all co-exist, but they often occur separately and appear at different stages, or any two of them may occur without the third. These different combinations, he concludes, can only be explained on the common etiological basis of antecedent syphilis. Erb warns against the error of regarding lymphocytosis as pathognomonic of central syphilis, and recounts two cases—one of glioma of the crus cerebri, the other of cervical carcinoma—in which lymphocytosis was well marked.

Lastly, he refers to the observations of Wassermann, Neisser, Plaut, and others on the sero-diagnosis of syphilis, by examination of the blood-serum or cerebro-spinal fluid. In syphilitic diseases a specific sero-diagnostic reaction is present, and specific anti-bodies, if demonstrated in the serum or cerebro-spinal fluid, are practically conclusive as to the syphilitic nature of the case. The almost constant presence of such anti-bodies in tabes and in general paralysis is strong confirmatory evidence of the syphilitic origin of these two affections. Unfortunately the technique of these observations is very complicated.

PURVES STEWART.

ON THE SYMPTOMATOLOGY OF ATROPHY OF THE OCCIPITAL

(76) **LOBE.** (*Zur Symptomatologie des atrophischen Hinterhauptslappens.*) A PICK (of Prague), *Arch. a. d. deutsch. psych. Univ.-Klinik in Prag*, 1908.

THE author reports the case of a man of seventy-five, with a characteristic senile mental disorder, and in whose physical condition apart from the ordinary senile changes and well-marked impairment of gait there was nothing to notice except a very interesting visual disorder. Post-mortem the brain showed a simple senile atrophy, which was especially well marked in the frontal and occipital lobes; there was no focal lesion in the more limited sense of the word.

The visual disorder consisted in frequent mistakes in the naming and perception of objects. There was no diminution of visual acuity, no disorder of colour sense, no limitation of the field of vision, no impairment of stereoscopic perception; the sensation of direction was also intact. There was no aphasia, but merely a slight verbal amnesia; no symptoms of apraxia were observed. The patient perceived quite well the part of an object in the centre of the field of observation, but was unable to synthesize it with the impressions from the periphery of the field into the perception of the total object. If the object were small the visual perception of it was possible; but if it were large, the patient made glaring mistakes. *e.g.* if shown a picture of a man's head larger than life size, he could name the eye or mouth correctly, but could not grasp the picture as a whole; if asked to point out certain features he would be correct with some, but with others would place them in quite absurd positions. He was very conscious of his inability, and frequently arrived at the correct conclusion by a process of reasoning rather than by one immediate act of perception. On the face of a large clock he lost his bearings, although he could immediately give a theoretical description of the position of the various hours. A second element in his disorder of visual perception was seen in his behaviour when an object, seen and recognised, was removed to a further distance or replaced by another; he persisted in groping for the distant object and in perseverating with the name of the first object.

The author considers that the disorder is in the function which composes the various elements of a visual picture into a unity, the combining faculty; he does not think that one is entitled to look upon it as a disorder of "secondary identification," a higher step in the hierarchy of the apperceptive processes. He correlates the disorder with the atrophy of the occipital lobe.

Probably many cases of so-called "mind-blindness" are really due to a more elementary disorder than the one usually assumed. The analysis of the psychological mechanism is interesting, and he

supports his views on the anatomo-clinical correlation by the short report of a second case with focal lesions in the occipital lobes. He sums up his views in the statement that there exist disorders of visual perception, which are due to an impairment of the synthesis of the corresponding visual impressions, and which may be opposed as a sensory ataxia to the better-recognised motor ataxia.

C. MACFIE CAMPBELL.

ON ASYMBOLIA AND APHASIA. (Ueber Asymbolie und Aphasie.)

(77) A. PICK (of Prague), *Arb. a. d. deutsch. psych. Univ.-Klinik in Prag*, 1908.

IN this review of the subject of asymbolia, Pick endeavours to clear the ground for the adoption of a uniform nomenclature. Finkelnburg had defined asymbolia as the disorder "owing to which the power either of understanding or of expressing concepts by means of acquired symbols is impaired." Under the influence of Kant's psychology he gave as illustrations of asymbolia impairment of recognition of the environment, of understanding religious and social forms.

These, however, belong to the sphere of complex mental elaboration, and Wernicke opposed such an extension of the application of the term. He limited the term to those disorders which affect the primary elaboration of the impressions of signs or sense-qualities of objects—optic, acoustic, etc.; the identification or recognition of the name of an object he separated from the identification of its other qualities or signs. He thus contrasted aphasia on the one hand with asymbolia on the other; asymbolia, thus limited to a receptive disorder, was practically the same as Freud's agnosia. Pick, calling attention to the broad biological principles of human adaptation to environment, shows that in the mental elaboration which is one link in any purposeful act of adjustment there may be both on the receptive and on the emissive side a hitch at that point, where the utilisation of symbols is a factor in the process; such symbols may be conventional or non-conventional. On these general grounds he gives the following classification of the disorders which may fitly be grouped under asymbolia:—

A. Emissive asymbolic disorders: 1. Motor-aphasic disorders of speech; 2. motor-agraphic disorders of writing; 3. motor amusia (musical expression);—(a) vocal, (b) graphic, (c) instrumental; 4. motor disorders of finger-speech (deaf and dumb language); 5. motor disorders of gesture; 6. disorders of expression by means of pantomime.

B. Receptive asymbolic disorders: 1. Sensory-aphasic disorders of speech; 2. sensory-aphasic, alexic disorders of writing;

3. sensory amusia (auditory and visual); 4. loss of the power of understanding the deaf and dumb language (?); 5. loss of the power of understanding gestures; 6. loss of understanding pantomimic actions.

The author fully realises the difficulty of a satisfactory classification in view of the many transition forms.

C. MACFIE CAMPBELL.

ON THE SIMULTANEOUS OCCURRENCE OF ACROMEGALY (78) AND SYRINGOMYELIA, WITH A DISCUSSION OF THE QUESTION OF THE OCCURRENCE OF ACROMEGALY WITHOUT CHANGES IN THE HYPOPHYSIS. (Ueber das gleichzeitige Vorkommen von Akromegalie und Syringomyelie. Zugleich ein Beitrag zur Frage nach dem Vorkommen von Akromegalie ohne Veränderung der Hypophysis.) KARL PETRÉN, *Virchow's Arch. f. Patholog. Anat.*, Bd. 190, 1907.

THE writer has observed a case of acromegaly which, from a clinical point of view, was quite typical, but post-mortem presented no sign of a growth in the pituitary fossa. The evolution of the disease was very slow; death was due to tubercular pericarditis.

The patient stated that both his grandfathers, as well as some other members of his family, had shown the same signs of acromegaly as he did. From a trustworthy source it was ascertained that a brother at least certainly had acromegaly. The writer has found from a study of the literature that a true hereditary acromegaly has already been recorded, but this is very rare.

At the autopsy certain anatomical changes characteristic of acromegaly were found. The bones of the cranium, however, were almost all very thin, and the foramina of the base of the skull were generally dilated. It is well known that in acromegaly the bones of the cranium are generally thickened, but in some cases the contrary condition has been found in some parts of the cranium, though not to such an extent as in this case.

There was a very high degree of bathrycephaly. This condition has already been noted, and is assumed by Launois and Roy to be characteristic of acromegaly; but, from an analysis of the literature, the writer demonstrates that this is not so.

In this case the hyphosis was not enlarged, and no microscopical changes of the gland were found, except a slight degree of sclerosis, which might arise from the age of the patient (50). The writer, after analysing all the published cases, finds that in a considerable

number of cases in which the diagnosis of acromegaly was quite certain, the hypophysis was not enlarged. The number of cases in which microscopical examination of the gland yielded negative results is not so great, but in recent years especially a number of such cases have been published (Dallemagne, Mitchell, Huchard, and Launois, etc.); consequently the doctrine of constant changes of the hypophysis in acromegaly must now be abandoned.

Examination of the spinal cord revealed an unexpected syringomyelia. This had given rise to no clinical symptoms. This is explained by the site of the anatomical changes which occupied the region of the central canal and the base of the anterior horns (the posterior horns being almost entirely free from the disease). The lesions were most extensive in the third and fourth dorsal segments, where the anterior horns were almost completely destroyed. (This, we know, would give rise to no clinical symptoms).

The syringomyelia had not the usual microscopical appearance, as a true gliomatous tissue was not found; the affected parts of the cord contained only epithelial cells similar to those in the central canal, and fibrous connective tissue, connected with a great number of markedly sclerosed blood-vessels. The central canal was enlarged throughout the greater part of the cord, usually in the form of a narrow transverse slit. In connection with the central canal were found regular bands of epithelial cells; but the latter formed for the greater part an irregular mass, without any evident tissue between the cells.

The author examined the cord from another case of acromegaly which had shown typical signs of a growth in the pituitary fossa. Operation by Horsley's method was unsuccessful on account of hæmorrhage. Here also pathological enlargement of the central canal was found, though slight in degree; also some sclerosis of blood-vessels in the cord.

The author has gone thoroughly into all the published cases of acromegaly in which the cord was examined, and has come to the following conclusions:—

That some changes, such as a slight degree of sclerosis in the columns of the cord, are of no importance;

That increase of the connective tissue in the cord, found in some cases, is pathological, but may be explained, like the changes so often found in other organs, as the result of the acromegaly itself;

That such nervous diseases as multiple neuritis or meningo-myelitis, present in isolated cases, must be regarded as accidentally coinciding with the acromegaly;

That the pathological increase of the epithelial cells of the central canal is so frequent that it cannot be regarded as merely

coincident, but must have a causal connection with the acromegaly. This conclusion is the more inevitable since in the cases of the writer, of Schulz, of Bassi, of Collier, and perhaps in other two, a true syringomyelia was present along with the acromegaly.

Schlesinger, in his remarkable work, states that true acromegaly is never noted along with syringomyelia. The writer's investigations show that this opinion must evidently be abandoned, and that in some cases there may exist a causal connection between the acromegaly and the increase of epithelial cells in the central canal.

The paper concludes with some theoretical considerations. The possibility is also mentioned that as the increase of the epithelial cells of the hypophysis may be accepted as the cause of the enlargement of so many parts of the body in the ordinary cases of acromegaly, so the increase of the epithelial cells in the central canal of the cord—in spite of their different embryological origin—may perhaps be the cause of the enlargement of the distal parts of one or more extremities, which is so frequently observed in syringomyelia.

AUTHOR'S ABSTRACT.

**ON THE SYMPTOMATOLOGY AND ANATOMY OF TUMOURS
(79) OF THE PITUITARY DUCT.** (*Zur Symptomatologie und Anatomie der Hypophysenganggeschwülste* [Erdheim].) E. STRÄUSSLER, *Arb. a. d. deutsch. psych. Univ.-Klinik in Prag*, 1908.

THE clinical and anatomical report of a case of tumour of the hypophysis. The patient was a man of 38, who for many months suffered from what was diagnosed as neuralgia of the fifth. After an X-ray examination, the diagnosis of tumour of the hypophysis was made. At this period, in addition to attacks of headache and vomiting, there was limitation of the temporal field of vision on one side; the knee-jerks and Achilles-jerks were absent, but on a later examination were elicited, although feeble. Trephining relieved the headache to a certain extent, but otherwise did not influence the course of the disease. Mentally the patient presented disorientation, confabulation, and impaired retention—Korsakoff's symptom-complex.

At the autopsy the hypophysis was found to be attached by a short stalk to a cystic neoplasm; microscopically this consisted of connective tissue with cavities, the walls of which were lined with pavement epithelium. The posterior columns of the cord showed some degeneration.

C. MACFIE CAMPBELL.

ACUTE ATAXIA. (Ueber acute Ataxie.) BREGMAN. *Deutsche*
(80) *Zeitsch. f. Nervenheilk.*, 1907, p. 409.

AFTER a discussion as to the various possible lesions in the cerebrum, cerebellum and peripheral nerves, capable of producing acute ataxia, Bregman records two cases of his own, the first apparently of cerebellar origin, the second, he considers, due to a combination of cerebral and peripheral disease.

Case 1.—An alcoholic woman aged sixty. Four weeks before admission she had sudden unconsciousness lasting about half an hour. Ever since she has been unable to walk, the speech has been unintelligible, and the movements of all the limbs inco-ordinate. Dry gangrene occurred in several fingers of the right hand, and the right radial artery was markedly sclerosed. There was static ataxia which gradually improved, though the patient retained the tendency to fall to the left side. Voluntary movements of all the limbs were inco-ordinate, especially on the left side, the left upper limb being most affected. Articulation was ataxic and explosive. There was nystagmus on lateral movement of the eyes, especially to the left. The deep reflexes were lost. The plantar reflexes were flexor in type.

Case 2.—A girl of fourteen. Sudden "unconsciousness" lasting a few minutes and followed by pain in the neck. (The patient ran a hair-pin into her scalp in falling.) On recovering consciousness she had total loss of power in both upper limbs, with total anæsthesia, the feet being well moved. There was high fever for several days, and the patient complained of pain on passive movement of the limbs. Speech and swallowing were unaffected. After a week she began to recover, but reeled in a drunken fashion. Movements reappeared in the upper limbs, commencing in the fingers. No headache or vomiting. On examination, the upper limbs were feeble and ataxic. There was no muscular atrophy. The patient could not feed herself. The lower limbs were strong and devoid of ataxia. There was slight blunting of sensation to touch in the fingers and loss of joint-sense in the hands. The knee-jerks and ankle-jerks were normal, and there was a pseudo-ankle-clonus. The plantars at first appeared to be extensor, but later were indefinite. The cranial nerves were normal. There was tenderness on pressure on the nerve-trunks. Bregman considers that the above symptoms can only be explained by a combination of a lesion of the peripheral nerves and a lesion of the brain. The diagnosis, however, is far from convincing, the most probable explanation, that of hysteria, not even being mentioned.

PURVES STEWART.

ON DISORDERS OF ORIENTATION ON ONE'S OWN BODY.(81) (*Ueber Störungen der Orientierung am eigenen Körper.*)A. PICK (of Prague), *Arch. a. d. deutsch. psych. Univ.-Klinik in Prag*, 1908.

THE patient was a woman of 55, with very marked memory defect and general mental impairment; apart from marked exaggeration of the knee-jerks there were no neurological symptoms of importance. A peculiar disorder of orientation was present. When asked to point out different parts of her body, she frequently was unable to do so, or only arrived at the part as if by a sudden intuition, after much inappropriate fumbling. When unable to point to her left eye, she said, "I don't know—I must have lost it." The author attributes her inability to localise parts of her body to deficient power of visual representation of the body or part specified. The fact that the patient would seek around for the part elsewhere than on her body is explained as follows by the author: The failure of the visual representation leaves the attention without any definite localising guide, and fixed by the verbal concept of the object sought. Similarly, the fact that, on seizing the nose of the examiner, she called it her own, depended on the absence of the visual picture of her own body.

C. MACFIE CAMPBELL.

NON-TRAUMATIC PSEUDOSPASTIC PARESIS WITH TREMOR.(82) (*Über pseudospastische Parese mit Tremor nicht traumatischer**Ätiologie.*) K. KRAUSE, *Monatsschr. f. Psychiat. u. Neur.*, 1907, Bd. 22, *Ergänzungsheft*, S. 54.

KRAUSE first reviews the previously published cases of this remarkable syndrome, first described by Fürstner and Nonne in 1896, and gives an account of the symptoms typical and atypical belonging thereto. He then describes two cases in which the syndrome developed from a hysterical pseudo-tabes and an alcoholic neuritis respectively. In neither case was trauma present, whereas in only two of the previous cases has this factor been absent. He discusses the nosological grouping of the syndrome, reckons it as hysterical in his two cases, but thinks it may possibly be hypochondriacal in others. He reaches the curious conclusion that the hysteria was of "toxic" origin in his two cases, and gives no psychological analysis of them.

ERNEST JONES.

CONSTITUTIONAL ASTHENIA. (L'Asthénie constitutionnelle.)

(83) PAUL LONDE, *Rev. de Méd.*, Nov. 1907, p. 1023.

LONDE gives a descriptive account of the various symptoms that may be called asthenic, and traces their evolution into "constitutional neurasthenia." He attributes them, without of course giving the least evidence for his opinion, to congenital cerebello-sympathetic deficiency. The treatment advocated is as vaguely based as the rest of the article.

ERNEST JONES.

ON ABASIA OR DYSBASIA. (Über Abasie resp. Dysbasie.) E.

(84) TRÖMNER, *Monatsschr. f. Psychiat. u. Neur.*, Bd. 22, Ergänzungsheft, S. 132.

THIS is a broad discussion of functional disturbances of gait. The author takes up Binswanger and Ziehen's position, which is as follows: The more restricted is the lower limb weakness to gait alone, *i.e.* the more specific is the symptom, the more certain is it to be of a functional nature. Pure abasia, *i.e.* disturbance confined to gait, is probably always hysterical. The cases in which the relation to gait predominates the disturbance, but in which weakness is also present under other conditions, the author appropriately terms dysbasia; this symptom occurs in hypochondria and neurasthenia. Apart from this is basophobia, an instance of the obsessional states. Four interesting cases are fully described here, and the grouping and diagnosis of them discussed, as well as that of "stammering gait," previously published by the author (see *Review of Neur. and Psychiat.*, 1906, p. 770).

ERNEST JONES.

DIFFERENTIAL DIAGNOSIS OF ORGANIC AND FUNCTIONAL

(85) **APHONIA. (Die differentialdiagnostische Bedeutung der organischen und funktionellen Aphonie.)** ERNST BARTH, *Deutsche Med. Woch.*, Nov. 28, 1907, S. 1999.

THIS is a general account of the subject in a short note that contains nothing new. The author lays stress on the frequency with which functional aphonia develops as a sequel to organic changes.

ERNEST JONES.

HYSTERICAL PURE WORD DEAFNESS. (Über hysterische reine

Worttaubheit.) A. KNAPP, *Monatsschr. f. Psychiat. u. Neur.*, Dec. 1907, Bd. 22, S. 5-36.

KNAPP relates the case of a woman of 35, who presented at first complete deafness on both sides, paresis of the left lower facial, with "hysterical" changes in character. The facts that the paresis was

confined to the face while no other signs of cerebral disease were present, and that the electrical excitability was normal, led to the diagnosis of hysterical paresis. This was confirmed by the disappearance of the symptom after a single suggestive application of faradism. After a fortnight's similar treatment the deafness gradually disappeared, leaving however a pure word deafness. This resembled subcortical sensory aphasia in that speech, reading, and writing were unaffected, and differed from it only in the fact that the patient could hear her own voice perfectly. The word deafness was improved so far as hearing female voices was concerned, but remained permanent for male voices. The treatment consisted in painful applications of electricity and in making threats of various punitive measures. To the author's evident surprise and resentment the patient's dislike of him did not diminish under treatment, nor did her refractoriness and inaccessibility, to which he naïvely attributes the failure of his treatment.

Knapp claims that this is the first case of the kind to be published. (The Reviewer may remark that this is far from being true; Calligaris, for instance, published a similar case of pure "subcortical" word deafness in hysteria in the Rome Otological Clinic Records for 1905.) Mann has published a case of "cortical" sensory aphasia in hysteria, and "transcortical" aphasia is often seen in Ganser's syndrome. "Subcortical" motor aphasia is of course well recognised in hysteria, but not the "cortical" type.

It is unfortunate that no psychological analysis of the origin and nature of the syndrome was made in this important case.

ERNEST JONES.

MECHANISM OF A SEVERE BRIQUET ATTACK AS CONTRASTED WITH THAT OF PSYCHASTHENIC FITS.

E. JONES (of London), *Journ. of Abn. Psych.*, Dec. 1907-Jan. 1908.

CERTAIN points in the differentiation of some of the psychoneuroses are taken up, and the author states that many such conditions have been, and still are, incorrectly diagnosed under the terms psychic epilepsy, grand mal, petit mal, etc. In making a diagnosis, evidence should be gained from observation of the mechanism initiating an individual fit, the character of the fit, and from the examination of the patient during the interval period.

Typical hysterical fits are less frequently mistaken than the grand or petit mal variety, for the reason that every symptom of a grand mal attack, from the fixed pupil to sphincter relaxation, may occur as well in functional affections; it is therefore almost impossible to exclude hysteria from the observation of the fit alone.

Again, it is necessary to differentiate hysteria from psychasthenia, and the author summarises some of the aspects of this problem by stating that "the disaggregation of hysteria is massive, while that of psychasthenia is molecular."

Primary hysteria is often complicated by psychasthenic symptoms; the reverse is very rare. C. H. HOLMES.

ANOREXIA NERVOSA IN CHILDREN. F. FORCHHEIMER, *Arch. (88) of Pediat.*, Nov. 1907, p. 801.

THE author reports four cases of the condition in children aged respectively seven years, twelve years, one year, and three years.

He believes that all the cases can be cured provided they are properly treated. "In order that a child be cured of it there are required a proper physician, a proper nurse, and proper surroundings."

A. DINGWALL FORDYCE.

SPONDYLITIS INFECTIONOSA FOLLOWING DENGUE. (Zur (89) *Kenntnis der Spondylitis infectiosa nach Dengue-Fieber.*) H. SCHLESINGER, *Arb. a. d. Neurol. Instit. a. d. Wien. Univ.*, Bd. 16, Teil 2, 1907, S. 13.

THIS paper deals with a condition which has not hitherto been described in connection with dengue, but which is well known as a sequela of enteric fever under the name of "typhoid spine." From a prognostic standpoint spondylitis infectiosa resembles many other non-purulent inflammatory processes in the nervous system following infectious disease, *e.g.* acute encephalitis, serous meningitis, myelitis, and polyneuritis. In most cases there is complete or, at least, considerable restoration of function.

A man, aged thirty-six, contracted dengue in Cairo. On the ninth day of the disease meningeal symptoms appeared, and lasted nineteen days. During convalescence weakness of the lower limbs and ataxia developed. Walking was only possible by the help of sticks. The muscles of both legs were considerably wasted, the knee and ankle jerks were exaggerated, and there was patellar and ankle clonus on both sides. Babinski's sign was absent, and there were no superficial or deep sensory changes except for meralgia paræsthetica and a zone of thermohyperæsthesia at the level of the iliac crest. The sphincters were unaffected. Examination of the vertebral column showed that the normal lumbar lordosis was replaced by a kyphosis involving all the lumbar spine, percussion of which was painful. The part was kept rigid, and unexpected movements of the trunk caused pain. The skin was not affected. The X-rays showed

subluxation and rotation of the second lumbar vertebra and an exudation between the first and second lumbar vertebræ. Tuberculosis was negatived by the absence of personal or family antecedents and a negative reaction to tuberculin. Intra-muscular injections of atoxyl and hot-air baths were prescribed. Gradual improvement took place, but the spondylitis lasted several months.

J. D. ROLLESTON.

THE PATHOGENESIS OF DIPHTHERITIC PARALYSIS AND
 (90) **HEART FAILURE IN DIPHTHERIA.** (*Zur Pathogenese der postdiphtherischen Lähmungen und des Herztodes bei Diphtherie.*) SPIELER, *Arch. a. d. Neurol. Instit. a. d. Wien. Univ.*, Bd. 15, Teil 1, 1907, S. 512.

AFTER reviewing the current theories as to the nature of diphtheritic paralysis, Spieler records the clinical history and post-mortem findings in three cases. The first case died suddenly of cardiac paralysis in the fourth week, three days after the occurrence of paralysis of the neck muscles. In addition to advanced fatty degeneration of the myocardium, histological examination revealed no lesions in the central nervous system, but considerable changes in the vagi and spinal accessory nerves, while the anterior crural nerves were found to be quite normal. In the second case, which died within the first week with symptoms of palatal and cardiac paralysis, the central nervous system was again found to be intact, while advanced changes were found in the palatine nerves and to a less degree in the two vagi. In the third case, which also died within the first week, clinically there were no premonitory cardiac symptoms, and the sudden cardiac failure came as a surprise. Examination, however, showed, in addition to degeneration of the myocardium, considerable signs of degeneration in the vagi. In all the three cases the degree of degeneration of the myocardium would hardly have sufficed to have produced so sudden, and in the last two cases so early, a death, whereas the advanced degeneration of the vagi readily explained this. The author concludes that sudden failure of the heart in diphtheria finds its explanation in the existence of early peripheral degeneration of the vagus. The peripheral neuritis is due to the direct action of the local diphtheritic process on the terminal branches of the nerves supplying the diseased area. This view is supported clinically not only by the usual course of diphtheritic paralysis, but also by the fact that in paralysis following cutaneous diphtheria, the structures adjacent to the diphtheritic lesion are first affected.

J. D. ROLLESTON.

- OBSTRUCTION IN THE NOSE OR IN THE THROAT AS CAUSE
(91) OF NERVOUS AND MENTAL DISEASES IN SCHOOL
LIFE.** R. H. JOHNSTON, *N.Y. Med. Journ.*, Nov. 30, 1907,
p. 1023.

THIS paper is merely a short résumé of the nervous mental disease resulting from adenoids, enlarged tonsils, deviated nasal septa, etc., in children, probably all more or less due to the consequent deficient oxygenation. Aproxexia, night-terrors, nocturnal enuresis, pseudomeningitis, outbursts of temper, and reflex nervous cough are among the conditions mentioned. J. H. HARVEY PIRIE.

- TRAUMA IN THE ÆTIOLOGY OF NERVOUS DISEASES.** (Der
(92) Unfall in der Ætiologie der Nervenkrankheiten.) KURT
MENDEL, *Monatsschr. f. Psychiat. u. Neur.*, 1907, Bd. 21, S. 468,
550; Bd. 22, S. 158, 264, 373, 544.

IT is impossible here to do more than indicate this remarkable contribution, eighty-four pages long, which resumes all our knowledge on the subject. The literature is considered fully, and criticised in detail. Only organic affections, chiefly general paralysis, tabes, tumour, and meningitis, are dealt with, and after every section a short list of conclusions is given. The whole article forms an invaluable contribution to our precise knowledge of this ætiological factor. ERNEST JONES.

- THE OPHTHALMO-REACTION TO TUBERCULIN: A RECORD
(93) OF 300 OBSERVATIONS ON CHILDREN.** (L'oculo-réaction
à la tuberculine en clinique infantile, d'après 300 observa-
tions.) J. COMBY, *Le Bulletin Médical*, 20 novembre, 1907.

PROFESSOR CALMETTE, the Director of the Pasteur Institute of Lille, described before the Academy of Science in June last a new test for the diagnosis of tuberculosis.

The ophthalmo-reaction to tuberculin, as it is called, is carried out by introducing into the eye a drop of a definite dilution of tuberculin. If the individual is not a tuberculous subject, nothing is noticed. On the other hand, if he is a subject of tuberculosis, whether active or latent, from five to ten hours, or, in rare cases, even as late as forty-eight hours, after the introduction of the drop, the eye begins to redden, and a more or less extensive conjunctivitis develops, which may last as long as eight or ten days.

Calmette employed small capsules, each of which contained five milligrams of dried tuberculin. To this ten drops of boiled

water are added. A single drop of this solution is introduced into the eye.

Facts are required to determine the exact value of the method. A number of corroborative observations have already been reported.

Comby is of opinion that, in the case of children, it is advisable to use a somewhat weaker dilution, and recommends a dilution of half the strength (twenty drops of water added), for on more than one occasion when using the dilution originally recommended by Calmette, he has seen a somewhat intense conjunctivitis produced.

When this dilute solution is employed three types of reaction may, according to Comby, be recognised.

1. A slight reaction which may escape notice, if one does not pay special attention to the angle of the eye and compare it with that on the opposite side. This reaction rarely persists longer than two or three days.

2. Reaction of moderate intensity. The inner half of the eye is injected, the caruncle stands out prominently, both the palpebral and ocular mucous membranes are congested. The reaction persists for from 5 to 7 days.

3. Intense reaction with general injection of all the conjunctiva. There may be œdema with occlusion of the lids, a purulent secretion, unpleasant sensations about the eye, and pain. At times vesicles are to be seen at the corneo-scleral junction. With a 1 in 200 solution this reaction is rare; it is not seen once in twenty cases and the prognosis need not cause anxiety.

Comby has never seen the slightest general reaction, a weighty point in favour of this test as opposed to the subcutaneous injection of tuberculin. The reaction may be repeated again and again at intervals of days or weeks, but the result remains constant. Children presenting a perfectly healthy appearance, who are affected with latent tuberculosis, give a positive reaction, as do those affected with all forms of active tuberculosis.

Among the children who had not shown the reaction, eight died and were examined post-mortem. In not one was there a trace of tuberculosis. Ten of those children who gave a positive reaction died and were examined post-mortem: tuberculosis was present in every case.

The results of those observers who have expressed doubt as to the value of the reaction may, Comby believes, be explained by differences in technique.

The reaction is contra-indicated in the presence of any local ocular condition which might be thereby intensified. The reaction has to be carefully looked for in slight cases, while it is

necessary to remember that in some cases it takes a long time to appear.

Comby concludes his paper with the following sentence: "The ophthalmo-reaction to the tuberculin is very simple, very elegant, and very sure. It has not deceived me once among the 300 healthy and diseased children on whom I have tested it."

EDWIN BRAMWELL.

ARTERIOSCLEROSIS. G. L. WALTON and W. E. PAUL (of Boston),
(94) *Journ. Amer. Med. Assoc.*, Jan. 18, 1908.

THERE is a general tendency at the present time towards a reaction from the somewhat widespread movement to attribute to arteriosclerosis all sorts of nervous symptoms; in fact, given the symptoms, it is almost conceded that arteriosclerosis must exist, although no sign appears in those arteries which are palpable. This position is somewhat fortified by the knowledge that arteries may be hardened in one part of the body and not in another.

With a view to throwing more light upon this somewhat obscure subject, the authors have examined a series of one hundred individuals presenting marked and obvious sclerosis of palpable arteries, to ascertain whether or not prominent nervous symptoms, commonly attributed to this condition, were present.

(1) Headache—occurred in only 22 per cent. The percentage is lower than that found in ordinary healthy individuals, if the young and middle-aged are included. In eye-strain, 69 per cent. of cases suffer from headache. The authors conclude that in the eye-strain of advancing years, headaches are sometimes relieved by the existence of arteriosclerosis.

(2) Vertigo—was found in 65 per cent.

(3) Apoplectiform attacks—were found in 34 out of the 100 cases. These may be fairly attributed to arteriosclerosis, although it must be remembered that the question is often complicated by the co-existence of renal involvement.

(4) Progressive loss of memory—was complained of in 48 cases.

(5) Insomnia—occurred in but 30, which would place it in the doubtful category.

(6) Irritability, anxiety, morbid fears, etc.—were present in 40 cases. In one-half of these the symptoms had been present throughout life, and had not increased of late.

Renal and cardiac changes were investigated, and blood pressure noted under varying conditions.

Signs of renal degeneration were present in 36 per cent. of the cases; cardiac enlargement in 86 per cent. of the cases in which

renal involvement was present; those without renal involvement showed 36 per cent. The average blood pressure in cases showing neither kidney disease nor cardiac enlargement was 147, taken with the Riva Rocci instrument; those with cardiac enlargement alone, 168; with kidney disease alone, 173; with both, 195. These facts show (1) that renal degeneration is the prominent factor in the production of cardiac enlargement; (2) that while either renal degeneration or cardiac enlargement is attended by moderately high blood pressure, the existence of both these conditions is accompanied by very high blood pressure.

The authors conclude that while arteriosclerosis is directly productive of apoplectiform attacks and of vertigo, and while it plays a part in the loss of memory as well as in the other failing powers of involution, it does not produce headache except as the immediate result of apoplectiform attacks. Arteriosclerosis may appear in elderly neurasthenics as in any other group of elderly persons, but further study of the relationship of the two conditions is desirable. Renal disease is a prominent factor in the cardiac enlargement often present in cases of arteriosclerosis. Arteriosclerosis without cardiac enlargement or renal disease is only exceptionally accompanied by a very high blood pressure. If cardiac enlargement or renal disease is present, moderately high blood pressure; if both are present, very high blood pressure is the rule.

C. H. HOLMES.

PSYCHIATRY.

A CONTRIBUTION TO THE STUDY OF HEREDITY. (*Beitrag* (95) *zur Lehre von der Heredität.*) A. PILCZ, *Arch. a. d. Neurol. Instit. a. d. Wien. Univ.*, Bd. 15, Teil 1, 1907.

IN this paper Pilcz tabulates and analyses 2000 cases of mental disorder, taken from the registers of the Psychiatric Clinic of Vienna University, the cases having a neuro- or psychopathic parentage (insanity, epilepsy, migraine, organic spinal diseases, drunkenness, suicide, apoplexy, or character anomalies), generally of pronounced form. From his investigation the author draws the following conclusions:—

1. The hereditarily transmitted predisposition to a mental disorder differs according to the special clinical form, quantitatively and qualitatively.

2. Quantitatively, cases of progressive (general) paralysis, senile and arterio-sclerotic dementia, and also to a certain degree the non-katatonic form of dementia præcox, show a much less marked neuropathic heredity than the other forms of insanity.

3. Qualitatively, and speaking in general terms, there are different predispositions; *i.e.* the hereditary factors differ according to the particular form of mental disorder in the descendant.

4. As to direct heredity, homogeneous heredity is the general rule; ascendants and descendants tend to offer the same clinical forms, a noteworthy exception to this rule being furnished by non-katatonic dementia præcox, in which the ascendants have not presented the same disease but, commonly, general paralysis. (Thus of 51 cases of non-katatonic dementia præcox, the parents exhibited in 7 imbecility, in 1 moral insanity, in 3 melancholia, in 2 amentia, in 3 periodic psychoses, in 1 a neurasthenic psychosis, in 3 alcoholism, in 2 dementia præcox, and in 25 paralytic dementia. *Rev.*).

5. Besides the *hérédité similaire* the following play the chief rôle in psychotic heredity:—Alcoholic insanities in all psychoses except the types instanced under two; the affective psychoses in melancholia and circular insanity; senile dementia in general paralysis, and simple weak-mindedness in dementia præcox.

6. Epileptic and alcoholic psychoses might be quantitatively included with the hereditary degenerative insanities but direct *psychotic* heredity plays in these two psychoses a minor part.

7. As regards other hereditary factors, alcoholism in the ascendants is important in imbecility, the alcoholic psychoses and epileptic insanity, with the latter also epilepsy and migraine. Suicide is found most frequently in the heredity of the affective psychoses—a further proof of the intimate connection between suicide and melancholia. Tabes dorsalis, when it occurs, is found most frequently in the heredity of hebephrenia and general paralysis (as is also general paralysis itself), and apoplexy in the parents of general paralytics, arterio-sclerotic dements and melancholics.

8. Within the group of alcoholic insanities direct psychotic insanity is most marked in alcoholic paranoia and the so-named states of pathological drunkenness (*pathologische Rauschzustände*), a marked feature being the great frequency of epilepsy and migraine in the parentage.

R. CUNYNGHAM BROWN.

THE CEREBRO-SPINAL FLUID IN PARESIS, WITH A SPECIAL

(96) REFERENCE TO ITS CYTOLOGY. W. B. CORNELL (of Baltimore), *Amer. Journ. of Insan.*, July 1907.

THE author draws the following conclusions from recent literature on the subject, and a series of twenty-five cases of his own which were punctured thirty-seven times.

1. All cases of paresis exhibit a spinal leucocytosis and increase of albumen. 2. This sign is also, from point of view of its con-

stancy, in all probability the earliest. 3. The diagnostic value of a negative puncture is often of greater value than a positive one. 4. The cell-counting method with Fuchs and Rosenthal's slide is more accurate and rapid than the centrifuge technique, and has the great advantage in permitting comparative results. 5. The use of Unna's polychrome blue in the mixing chamber permits a simultaneous differential count. 6. A differential count is important in differentiating the paretic fluid from others, especially where the cytosis is due to a small number of polynuclears. 7. The conditions under which syphilis produces a spinal leucocytosis demand further investigation, especially regarding the number and character of the cells. The increase of cells in the paretic fluid is apparently independent of any long antecedent syphilis. 8. There seems to be a correlation, both qualitative and quantitative, between the spinal and hæmic leucocytosis, which particularly refers to the mononuclears, but includes the polymorphonuclears, especially after convulsions.

C. H. HOLMES.

FURTHER INVESTIGATION ON THE GALVANIC PHENOMENON AND RESPIRATION IN NORMAL AND INSANE INDIVIDUALS. (97) C. RICKSHER (of Hawthorne, Mass.) and C. G. JUNG (of Zürich). *Journ. of Abn. Psych.*, Dec. 1907, Jan. 1908.

THE authors reach the following conclusions:—

(1) The galvanic reaction depends on the attention to the stimulus and the ability to associate it with other previous occurrences. This association may be conscious, but it is usually subconscious.

(2) Physical stimuli as a rule cause greater galvanic fluctuations than do the psychical in the experiments. This may be due to the fact that they occurred before the psychical, early stimuli nearly always causing greater reactions than do later ones.

(3) While the normal reactions vary greatly in different individuals, they are as a rule always greater than pathological reactions.

(4) In depression and stupor the galvanic reactions are low, because the attention is poor and associations are inhibited.

(5) In alcoholism and in the euphoric stage of general paralysis the reactions are high, because of the greater excitability.

(6) In dementia the reactions are practically nil, because of the lack of associations.

(7) The reactions show great individual variations, and within certain rather wide limits are entirely independent of the original bodily resistance.

The pneumographic results may be summarised as follows:—

(1) The inspiratory rate varies according to the individual, and no general rule can be given.

(2) The amplitude of the inspirations is generally decreased during the rise of the galvanic curve.

(3) The decrease in the amplitude, however, has no relation to the height of the galvanic curve, but varies according to individuals.

(4) In cases of dementia, where there is no galvanic reaction, the changes in the respirations exist, but are very slight.

C. H. HOLMES.

PATHOLOGY AND TREATMENT OF OBSESSIONAL STATES.

(98) (*Zur Pathologie und Therapie der Zwangsneurose.*) WOLFGANG WARDA, *Monatsschr. f. Psychiat. u. Neur.*, Bd. 22, *Ergänzungsheft*, S. 149.

THIS paper is based on some of Freud's earlier views on the subject. Warda maintains that obsessional states are always due to the suppression of various sexual events in infancy, but gives no description of the pathology of the condition beyond the simple statement of this fact. He relates four not very convincing cases, which are poorly analysed; he seems not to be aware of Freud's later work on symbolisation, etc.

ERNEST JONES.

CLIMACTERIC PSYCHOSES. (*Über die Psychosen des Klimakteriums.*)

(99) HANS BERGER, *Monatsschr. f. Psychiat. u. Neur.*, 1907, Bd. 22, *Ergänzungsheft*, S. 13.

AFTER a few references to previous writings on the subject, Berger gives a short account of the material of the Jena clinic during the past nine years. Of 326 female patients between the ages of 40 and 55, in only 14 had the psychosis developed actually during the climacteric period (organic affections being excluded). Two of these cases are briefly described, and the general conclusions of the others given. In 10 of the 14 melancholia was the form present. Eight cases recovered completely, two committed suicide, and four ended in dementia.

ERNEST JONES.

ALCOHOLIC INSANITIES. A. GORDON (of Philadelphia), *Journ. (100) Amer. Med. Ass.*, Nov. 16, 1907.

THE author bases his paper on the study of four hundred and thirty-seven cases, and draws the following conclusions. Alcoholic insanity presents special characteristic features, which are not difficult as a

rule to distinguish from other analogous conditions. Acute cerebral alcoholism presents three states—delirious, confusional, and stuporous. The intensity of these states varies according to whether a sub-acute form or a form with delirium tremens is dealt with.

The chronic form invariably leads to dementia. In the development of the latter form, delusions, hallucinations, and illusions may or may not manifest themselves.

Symptoms of other psychoses may confuse the picture, but close observation will make the proper interpretation possible. Symptoms of cerebral alcoholism may be superimposed upon other psychoses, but it does not follow that alcoholism causes these psychoses.

The conception of alcoholic melancholia, mania, paranoia, or alcoholic general paralysis is unscientific, as it is not based upon accurate observations.

The paper contains very little that is new, and the reference to alcoholic melancholia, mania, and paranoia is vague, for it is certain that the depressed, excited, and delusional forms of alcoholic psychoses are comprehensive and well recognised.

C. H. HOLMES.

A CASE OF PERIODIC MELANCHOLIA, COMBINED WITH
 (101) **HYSTERIA AND TABES DORSALIS WITH PECULIAR**
ATTACKS OF MIGRAINE. (Ueber einen Fall von periodischer Melancholie, kombiniert mit Hysterie und Tabes dorsalis, mit eigenartigen Migräneanfällen. Zugleich ein Fall von akuter Veronalvergiftung.) M. PAPPENHEIM, *Arb. a. d. deutsch. psych. Univ.-Klinik. in Prag*, 1908.

THE patient, a young man with extremely bad heredity, who had suffered from attacks of migraine since boyhood, first came under observation at the age of 30. He had, at the age of 14, had fainting attacks; at the age of 20, after influenza, he was languid, sleepless, and without appetite, took pleasure in nothing. He later had several attacks of depression, and attempted suicide. On admission to the clinic he was a typical case of melancholia simplex, the melancholic phase of manic-depressive insanity (Kraepelin); in the physical condition there was nothing abnormal to note. Ten years later, in 1905, he returned to the clinic, having in the interval had several attacks of depression, and contracted syphilis in 1898. He now showed tabetic symptoms—lancinating pains, pupillary anomalies, lymphocytosis of the cerebro-spinal fluid, bladder disorders, sensory disturbances, Romberg's sign; certain elements in the clinical picture appeared to be of functional origin. The author describes in detail the attacks of migraine,

which began with auditory phenomena; he considers that on the basis of an inherited migraine were superimposed tabetic "acoustic crises," due to affection of the cochlear nerve. On both sides there was evidence of organic disease of the eighth nerve of a tabetic character. The anatomical basis of the attacks is discussed in detail.

The patient attempted suicide by taking $8\frac{1}{2}$ grammes of veronal, and the symptoms following on this attempt are recorded.

C. MACFIE CAMPBELL.

THE CRIMINAL RESPONSIBILITY OF INSANE PERSONS.

(102) MORTON PRINCE (of Boston), *Journ. Amer. Med. Ass.*, Nov. 16, 1907.

THE author reviews somewhat in detail the history of the laws governing irresponsibility in crime, states that the law of to-day was defined by English judges in 1843 in answer to inquiry by the House of Lords, and has continued to stand both in England and America to the present time. This law is not statute but common law. He quotes the questions and answers upon which the law was founded, and makes pertinent comments on conclusions drawn. For example, one of the answers most frequently quoted is as follows:—"To establish a defence on the ground of insanity it must be clearly proven that at the time of committing the act the accused was labouring under such a defect of reason, from disease of the mind, that he did not know the nature and quality of the act he was doing, or, if he did know it, that he did not know that he was doing what was wrong." The question, to which this answer was given, referred to the insane who were suffering from delusions, while the answer has ever since been applied to all insane whether they suffered from delusions or not. The phrase, "persons who labour under partial delusions only and are not in other respects insane," is also well criticised, as being a psychiatric assumption which probably has no basis in fact.

Prince concludes that the law to properly determine responsibility should be statutory and not common law; he approves of the system now employed in the States of Maine, New Hampshire, Vermont, and Massachusetts, when the defence of insanity is raised in criminal cases. Here the accused person is committed to an asylum and placed under the continuous observation of impartial experts until the question of his sanity has been determined. The Court abides by this decision, believing that medical insanity constitutes legal irresponsibility.

Some such system would be a marked improvement on the present thoroughly rotten system of expert testimony which exists rather universally in America.

C. H. HOLMES.

MENTAL ALIENATION AND CRIME. (*La Alienacion Mental y (103) el Delito.*) By JOSÉ INGEGNIEROS, *Arch. de Psiq. y Crim.*, Sept.-Oct. 1907.

PROF. INGEGNIEROS comments upon a case of a prisoner who had made a homicidal assault on his wife. Prisoner was an inveterate alcoholic with frequent crises of intoxication, followed by impulsive and anti-social conduct. Sentenced to two and a half years' penal servitude, he was placed in the prison infirmary for mental observation, where he was found to have delusions and auditory hallucinations, and was therefore transferred to the pavilion for criminal lunatics. As in the former case, Prof. Ingegnieros argues that his condition should have been discovered at the trial and the prisoner sent direct to the criminal lunatic asylum as a dangerous and irresponsible person.

R. CUNYNGHAM BROWN.

THE LIBERATION OF CRIMINAL LUNATICS. (*Liberacion y (104) Abandono de Alienados Delincuentes.*) By JOSÉ INGEGNIEROS, *Arch. de Psiq. y Crim.*, Sept.-Oct. 1907.

IN a short paper Prof. Ingegnieros of Buenos Aires University relates the case of a man, the subject of systematized delusions of persecution, who, under the influence of his delusions, committed a serious crime. The account given by the author puts in a very unfavourable light criminal procedure in Brazil. Notwithstanding abundant evidence of insanity, the accused was sentenced to a term of imprisonment. Whilst in prison it was discovered that the prisoner had committed murder a year previously, and also that he was insane. Taking a retrospective view of his crimes, the authorities pardoned the prisoner and put him at liberty, prior to his committal to an asylum. Prof. Ingegnieros criticises the numerous juridical errors in the case, and maintains :—

1. The original crime of the accused was that of a lunatic, and ought to have suggested from the first his state of alienation.
2. Once put on trial his intense delusional manifestations ought to have put in motion an expert inquiry, which would have resulted in his detention permanently as a dangerous lunatic.
3. Once condemned, his insanity ought not to have brought about his pardon but a revision of the process.
4. The pardon ought not to have been accompanied by an order for his liberation.

R. CUNYNGHAM BROWN.

THE INSANE AND THE PENAL LAW. (*Los Alienados y la Ley Penal*.) By JOSÉ INGENIEROS, *Arch de Psiq. y Crim.*, Sept.-Oct. 1907.

PROF. INGENIEROS furnishes here a still more striking example of the inadequacy of Brazilian legal procedure with regard to insane persons accused of crime. On the one hand, he says, lunatics who have committed serious crimes are sent by the intermediation of the police to ordinary asylums, and, on the other hand, insane but inoffensive, or, at least, not dangerous prisoners, are confined in criminal lunatic asylums or the lunatic wings of prisons—that is, that the criminal lunatic asylum or the lunatic wing of a prison is simply a judicial depository for the insane, and not, as he contends they should be, houses of detention for dangerous lunatics. Under the existing Brazilian law, he says that even homicidal lunatic prisoners may be set at liberty to the great danger of the public. The example given is that of a labourer who, under the influence of delusions of persecution, killed one man and wounded several others. Whilst in prison he received hallucinatory revelations from God and the Virgin Mary, and had delusions of persecution and of grandeur. Later he had visual hallucinations, and in the third stage of progressive systematized delusional insanity, after an incarceration of twenty months, was liberated. Shortly after this liberation, however, he travelled to Buenos Aires, and proclaimed himself in the Cathedral to be the Messiah. He was thereupon arrested, though with difficulty, by the police and consigned to the asylum under Prof. Ingenieros, who gives full clinical notes of the case.

R. CUNYNGHAM BROWN.

TREATMENT.

INFLUENCE OF MERCURIAL TREATMENT OF SYPHILIS (106) ON THE OCCURRENCE OF METASYPHILIS OF THE NERVOUS SYSTEM. (*Hat die Hg-Behandlung der Syphilis Einfluss auf das Zustandekommen metasypilistischer Nervenkrankheiten?*). PAUL SCHUSTER, *Deutsche Med. Woch.*, Dec. 12, 1907, S. 2083.

SCHUSTER gives a most valuable discussion, supplemented by important original observations, on this question. After reviewing the literature he states the results of personal inquiries as to previous mercurial treatment in 235 cases of syphilis of the nervous system, in 186 of which the presence of syphilis had been proved, and positive

answers as to treatment obtained; of the 235, 90 were cases of tabes, 45 of general paralysis, and 100 of lues cerebri. A far higher percentage had been thoroughly treated than in Neisser's collected series of 445 tabetics. He concludes, on these and other grounds, that (1) the clinical picture of metasymphilis is the same, whether there had been previous mercurial treatment or not. (2) There is no relation between the date of onset of these diseases, and the question of former treatment. (3) There is no evidence that mercurial treatment of syphilis has any preventive action in this connection. (4) There is some reason to suppose that metasymphilis may be due to syphilitic antibodies rather than to the syphilis toxin itself. He further mentions that mercurial treatment has no effect on anti-bodies present in the blood of syphilitics.

ERNEST JONES.

THE SURGICAL TREATMENT OF TUMOURS OF THE SPINAL

(107) **MEMBRANES.** (Die chirurgische Behandlung der Rückenmarkshautgeschwülste.) BRUNS, *Deutsche Zeitschr. f. Nervenheilk.*, Bd. 30, H. 5-6, 1907, S. 355.

IN his introductory remarks the writer emphasizes the importance of general and segmental diagnosis, knowledge of the nature of the tumour, and its influence on the cord, its roots and coverings. Intradural tumours only are considered. These are not so common as extradural, and are usually not so extensive, and usually primary and benign, extradural being often metastatic and malignant. Those of practical importance are various fibromata, fibrosarcoma, and other forms of sarcoma, including psammoma. The majority are situated in a lateral and posterior direction from the cord, many directly posterior and in the median line. According to Schlesinger, if symptoms of tumour of the cord appear in people over forty years of age, a benign form may be assumed. He also states that benign tumours are more frequent in the dorsal region, whilst malignant are more often seen in the cauda equina. Softening of the cord is exceptional. In most instances there is only compression, with long preservation of the axis cylinders, and a possibility of recovery of the functions of the cord after removal of the tumour. Inflammation of the cord and membranes is rare in true tumour, and nerve roots in its vicinity may even remain uninjured anatomically for a long time. This applies especially to those originating in the cord above the level of the tumour, distinct symptoms occurring in the region of those involved near their exit from the cord. The bone symptoms are in most cases limited to susceptibility to pressure on the spines and the bones above and below them. In typical cases isolated root

symptoms are first observed, then Brown-Séquard paralysis, later paraplegic symptoms, subsequently associated with distinct bone symptoms. In lesions of a number of anterior roots atrophic muscular paralyses occur, but these are only distinctly recognisable in the cervical, lumbo-sacral and lower dorsal cord, and the reaction of degeneration may be absent. If the cord itself is involved anæsthesia is more pronounced at the level of the lesion, and there are symptoms of interruption of conduction in all parts below it. In exceptional cases few of the classical symptom-complex remain, individual symptoms being absent or very insignificant, and the sequence varying considerably. Intermissions and disappearance of pain, and its entire absence throughout the whole course of the condition, have also been observed, notably in tumour of the dorsal portion. Intermission may occur in the periods between complete destruction of one root and involvement of another, or conduction may be gradually interrupted owing to compression of the cord. Total absence of pain may be due to the tumour producing complete interruption of conduction before irritating a root, or to early operation on a tumour, which if allowed to grow would ultimately have caused pain. In cases in which the pain radiates into distant nerve provinces from the beginning, it is probably projected, owing to compression of the cord. In some cases unilateral symptoms have not been observed in the early stage, possibly because they were present for so short a time, or owing to ischæmic softening, œdema or inflammation rapidly injuring the entire transverse section of the cord. There may also be considerable variation in the duration and sequence of the individual symptoms, but in all doubtful cases recent experience makes it clear that there should be no hesitation in recommending operation if the trouble is progressive, and notably painful. As regards the diagnosis, marked bone symptoms are in favour of caries, and also the existence of burrowing abscess. In carcinoma and sarcoma the demonstration of a still present or previously operated upon growth in another situation makes the diagnosis clear, but in doubtful cases operation should be advised, and the X-rays may be of service. The difficulties in distinguishing between intradural and intramedullary growths may be insurmountable. In intramedullary, unilateral root and Brown-Séquard symptoms appear simultaneously, while in intradural the former may be isolated for a long time, though atypical cases must be borne in mind. Intramedullary tumours also increase rapidly in the long axis of the cord. Extensive root or cord symptoms, with intense pain on coughing or sneezing, are in favour of extradural tumour, though these signs cannot be absolutely depended upon. In chronic myelitis the distinction can be made from a syphilitic anamnesis, if present. In doubtful cases an exploratory operation is justified. Confusion may arise in the later

stages of hypertrophic cervical pachymeningitis, if the course of the case is not closely observed. Circumscribed gummatous meningitis may exhibit all the characteristic symptoms, showing the advisability of trying antisyphilitic treatment before having recourse to operative procedures. In syringomyelia the symptoms are marked in the long axis of the cord. Pronounced trophic disturbances, severe curvature, long course and considerable remissions are in favour of this diagnosis. In multiple sclerosis the characteristic ocular symptoms are of material assistance, as is puncture in meningitis serosa spinalis circumscripta. As regards segmental diagnosis the writer refers to the difficulty in distinguishing between tumour of the cauda equina and lumbo-sacral region. The roots and their segments having similar functions in these regions, it is obvious that there can be no essential difference in the root and cord symptoms. In tumour of the cauda the symptoms are often bilateral from the beginning, and the pain is of special intensity, duration and extent, while reaction of degeneration is marked. Tumour in this region is also frequently malignant and diffuse. In addition to the segmental diagnosis it is necessary to acquire accurate information as to the longitudinal extent of the tumour. There may be oedema, inflammation of the cord and membranes, possibly meningitis serosa circumscripta, around the tumour, and if they extend above it the upper extremity of the tumour may be assumed to lie at too high a level. Sherrington's law is of service in this connection. In general the highest segmental symptoms of the tumour must be referred to the highest root region under consideration, the level of the root being taken at its exit from the cord, and not at its exit from the column. In typical cases the general and segmental diagnosis can usually be made with a moderate degree of certainty when, after more or less long persistence of root symptoms, cord symptoms appear. If the former are absent, the segmental diagnosis cannot be made until there are symptoms of considerable compression of the cord. In relation to the nature of the growth, the age of the patient and the position of the growth must be considered. The writer concludes that intradural tumours in general are favourable objects for surgical treatment, pathologically, anatomically, as regards their position, size, shape, and influence on the cord and roots. He refers to the brilliant results in this region as compared with those obtained in the brain, notably that in a large proportion of cases the cord recovers after severe injury and interference with function. He points out that although the operation is not so dangerous as trepanation of the skull, with extirpation of tumour, it is yet not free from risk.

DONALD ARMOUR.

**A BRIEF REPORT OF FURTHER EXPERIENCES IN THE USE
(108) OF PARATHYROID GLAND FOR PARALYSIS AGITANS.**

W. N. BERKELEY, *N.Y. Med. Journ.*, Nov. 23, 1907, p. 974.

DISEASE of the parathyroid has been put forward by the author and others as the origin of paralysis agitans. Autopsy findings are however, as he admits, conflicting, and give as yet little support to the theory. He has given ox parathyroid both hypodermically and by the mouth, in a form prepared by himself and found to be active when tested on parathyroidectomized dogs, to a considerable number of cases with progressive benefit. The benefit consisted in diminished rigidity, lessened pain, salivation cured (salivation is, he says, a common though little known symptom), shaking diminished or cured, voluntary control of muscles increased, and restlessness and insomnia nearly or quite abolished. From a week to three months may be required for the good effects to become manifest; even advanced cases have been very much improved. The treatment seems to be at least as satisfactory as, and more permanent than, any other yet suggested, and the "antispasmodic" action of the gland substance might prove symptomatically useful in other conditions.

J. H. HARVEY PIRIE.

**THE METHODS AND TECHNIC OF THE DEEP ALCOHOL
(109) INJECTIONS FOR TRIFACIAL NEURALGIA.** D'ORSAY

HECHT (Chicago). *Journ. of Amer. Med. Assoc.*, Nov. 9, 1907.

EXCISION of the Gasserian ganglion is followed by brilliant and apparently permanent results in cases of intense and persistent facial neuralgia; but it must be admitted that the operation is a difficult one, and even in the hands of its most skilled exponents is attended with a definite percentage of fatalities. Obviously any method which minimises the risk to life, if equally efficacious, will be welcomed. Schlösser, in 1900, suggested injections of alcohol in the treatment of neuralgia. He is at present engaged in the preparation of a treatise which will include all his observations. Hecht considers the subject under the following headings:—
1. Anatomic considerations; 2. Methods of (a) Schlösser, (b) Ostwalt, (c) Levy and Baudouin; 3. Laboratory and clinical observations (personal); 4. The needle, syringe, and solution; 5. Technic; 6. Prognosis.

The impression derived from a perusal of his paper is that the technic in the case of the trigeminal nerve is one necessitating very considerable manipulative dexterity and experience. The final conclusions of Schlösser, who admits that the cure is not permanent, although it persists for about a year, will be awaited with interest.

EDWIN BRAMWELL.

THE TREATMENT OF CHOREA MINOR. D'ORSAY HECHT
(110) (Chicago). *Illinois Med. Journ.*, Nov. 1907.

THIS is a record of the author's personal experience and that of others as to the treatment of chorea. He concludes from his own material, that "cases of minor chorea of moderate severity show no great difference in their duration, whether under a treatment entirely expectant or strenuously medicinal," and holds that "the appropriate treatment for chorea comprehends (1) rest and isolation; (2) improved hygiene and nutrition; (3) drugs judiciously used."

Interesting results are those of Wall,: (1) 29 cases treated with salicylate of soda—15 were well in less than three months, 14 in less than two months; (2) 38 cases treated with aspirin—35 were well in less than two months, and 3 in less than three months; (3) 165 cases treated with arsenic in various forms—114 were well in less than three months, and 63 in less than two months; (4) 27 cases treated with general measures and cod-liver oil—19 were well in less than three months, and 13 in less than two months. For a child 10 to 12 years old, aspirin may be given in doses of 15 grains, three times a day; for a child between 6 and 8 years, a powder containing 10 grains. Wall prefers to give it stirred up in cold milk on a full stomach.

EDWIN BRAMWELL.

POSSIBLE PROGRESSIVE GROWTH IN MUSCULAR EFFICIENCY AFTER FIFTY YEARS OF LIFE WITHOUT SYSTEMATIC PHYSICAL EXERCISE. H. FLETCHER, N.Y.
Med. Journ., Nov. 30, 1907, p. 1005.

IN this article, Mr Fletcher recounts his personal experiences; how, from being a physical wreck in the early forties, he became able in a few years to do the work of trained athletes, without marked evidence of over-exertion. Some years later still he was put through a series of severe tests by Professor Chittenden, when it was found that there was a progressive improvement in his physical efficiency and power of endurance. One does not, however, acquire much detailed knowledge of "Fletcherism" from this paper, as only the main principles of his mode of life are given. Eat what you like, but eat it with mental calm and appreciation, and with careful and thorough buccal treatment—this way lies the road to health, happiness and efficiency.

J. H. HARVEY PIRIE.

Reviews

ARBEITEN AUS DEM NEUROLOGISCHEN INSTITUTE AN DER WIENER UNIVERSITÄT. (*Festschrift zur Feier des 25 jährigen Bestandes des Neurologischen Institutes.*) Herausgegeben von Dr OTTO MARBURG., Bd. xv. u. Bd. xvi., Franz Deuticke, Leipzig und Wien, 1907, M. 25 jeder Band.

NEUROLOGISTS of every country will welcome the appearance of the *Festschrift* in commemoration of the twenty-fifth year of the Neurological Institute of the University of Vienna, and the tribute of appreciation and gratitude to its director, Professor Obersteiner, by his pupils. The editor, Dr Otto Marburg, Assistant at the Institute, opens the volume with a contribution to the history of the Institute. When it was founded in 1882 by Professor Obersteiner, two of his first pupils were Dr Beevor, now President of the Neurological Society of Great Britain, and Dr Boothe of America. The former contributes to the volume a paper on a case of pseudo-bulbar paralysis with complete loss of voluntary respiration.

How inspiring the work of Obersteiner has been is well known in a general way to everyone, but it is brought home to us by the fact that more than one hundred and fifty works have been published by his pupils, nearly all of which are substantial contributions to the progress of neurology.

We wish the distinguished President of the Institute a long life to continue his own investigations and to stimulate many others to reap where he has sown.

Abstracts of a number of the papers in these volumes will appear in this and subsequent numbers of the *Review*.

ALEXANDER BRUCE.

ARBEITEN AUS DER DEUTSCHEN PSYCHIATRISCHEN UNIVERSITÄTS-KLINIK IN PRAG. Edited by ARNOLD PICK (of Prague). Berlin, Karger, 1908. Pp. 143, with numerous illustrations in the text and 11 plates. M. 8.

THIS volume contains four articles by Pick, and three articles by other workers in the clinic; the former writes on disorders of orientation on one's own body, circumscribed senile brain atrophy as an object of clinical and anatomical study, asymbolia and aphasia, the symptomatology of atrophy of the occipital lobe. Fischer describes a patchy atrophy of the medullated nerves in the cortex of general paralytics; Sträussler discusses the symptomatology and anatomy

of tumours of the hypophysis; Pappenheim reports a case of a tabetic with periodic melancholia. The plates which illustrate the various articles are excellent.

Abstracts of the separate contributions will be found elsewhere in this number of the *Review*. C. MACFIE CAMPBELL.

DIE PROGRESSIVE ALLGEMEINE PARALYSE. 2nd Edition.

By v. KRAFFT-EBING, revised and enlarged by H. Obersteiner.
Alfred Holder, Vienna, 1908, pp. 217. Price M. 5.20.

THIS book, although based on the presentation of the subject by v. Krafft-Ebing in the first edition, has been so thoroughly revised that it may be regarded almost as a new work. Especially in the chapters dealing with the pathological anatomy and the pathogenesis is seen the progress of our knowledge of the subject made since the publication of the first edition. Scarcely one quarter of the original work has been incorporated unchanged in this edition. The literature of the subject receives special attention, and the bibliography at the end contains 549 references. The book is a good example of that clearness of presentation which characterises Obersteiner's publications, and contains the results of his own wide experience in addition to that of the author of the first edition. A sober criticism is given of the numerous modern theories with regard to the disease, and no better general presentation of the subject could be put in the hands of the student or practitioner.

C. MACFIE CAMPBELL.

THE BORDERLAND OF EPILEPSY. Sir WILLIAM R. GOWERS, M.D., F.R.S. J. & A. Churchill, 1907. Price 4s. 6d. net.

THE difficulty in diagnosis presented by anomalous cases resembling epilepsy is very great. For many years Sir William Gowers has kept a special list of all cases which seemed to be on the borderland of epilepsy, "near it, but not of it." In the present volume he sums up the views he has arrived at from a study of his very large material, and thereby adds another important contribution to a subject which his writings have already done so much to elucidate,

The subject-matter of the book has for the most part appeared in a series of lectures, abstracts of which will be found in last year's volume of this review. Faints, vagal attacks, vertigo, migraine, sleep symptoms, and their treatment are dealt with in successive chapters in this volume, which will be read by all who attempt to keep abreast of modern neurology.

EDWIN BRAMWELL.

NOTWENDIGE REFORMEN DER UNFALLVERSICHERUNGSGESETZE. (Necessary Reforms of the laws regulating accident insurance.) A. HOCHÉ (of Freiburg), Halle, C. Marhold, 1907, pp. 27. Price 75 Pf.

THIS brochure consists essentially of the communication on the subject made by Hoche in 1907, to the summer meeting of neurologists and alienists in Baden-Baden. In 1891 the subject had also come up for discussion, but at that time it was a question of defining clinical pictures and estimating the value of symptoms; in the present contribution the centre of interest has progressed from questions of diagnosis to that of therapeutics. The author talks of a popular epidemic of traumatic neuroses in obvious relation to the laws dealing with accident insurance in Germany.

His main conclusions are as follows :—A large proportion of the cases of traumatic neurosis are due to the accident insurance laws. These cases, although essentially curable, owing to the existence of the present laws very frequently do not recover. An examination of the causes at the bottom of the development of such disorders suggests definite means of limiting the number of such cases. The suggested means are : (a) the elimination of all avoidable mental irritants in the compensation proceedings; (b) the institution of special bureaux which would endeavour to provide suitable work for the patient, the work being in itself a valuable therapeutic agent; (c) the substitution, as far as possible, of the payment of a definite sum for that of a weekly or monthly allowance.

C. MACFIE CAMPBELL.

UN MOUVEMENT MYSTIQUE CONTEMPORAIN: Le Réveil religieux du Pays de Galles (1904-1905). J. ROGUES DE FURSAC. Paris: F. Alcan, 1907. Price, 2.50 fr.

THE documents dealing with the psychology of religious phenomena are often of such a date that they are difficult to use for scientific purposes. The lives of the saints are full of interesting features, a further analysis of which is impossible owing to insufficient data. It is therefore extremely instructive to have a scientific account of a contemporary religious revival from the pen of a competent alienist. It is probable, however, that a thorough analysis of the mental experiences of a few of those who took an active part in the revival would have proved of more psychiatric value than the general description of the movement *en masse*. The author does not confine himself strictly to an analysis of the special phenomena

of the revival; he takes some pains to sketch the environment in which the whole movement developed, of which it was an organic manifestation. From the great upheaval of 1735 there has been a constant series of revivals in Wales, and this spasmodic character of their mysticism is doubtlessly deeply rooted in the Welsh temperament. The Welsh have lived in intellectual retirement, and as the modern spirit penetrates the mass of the people the conditions necessary for such revivals will disappear.

The good effect of the revival was seen in a distinct elevation of public morality, especially with regard to the use of alcohol; on the other hand, certain unstable natures were unable to support the mystical exaltation, and the number of cases of psychoses with distinct religious colouring was increased.

The book is written in a pleasant style, and if it does not give any profound analysis of the phenomena, it at least gives a useful outline of an important contemporary movement.

C. MACFIE CAMPBELL.

AERZTLICHES UEBER SPRECHEN UND DENKEN. Von G. ANTON (of Halle-a-S.). Halle: C. Marhold, 1907, pp. 20.

THIS brochure consists of a general lecture delivered on the subject of disorders of speech and of disorders of thought closely associated with interference with the speech mechanism. The place which speech plays in ordinary thought is very clearly presented, and the subject is treated in a very suggestive manner.

C. MACFIE CAMPBELL.

BOOKS AND PAMPHLETS RECEIVED.

Frugoni. "Intorno ai Rapporti tra Pneumogastrico e Funzione Renals." *Soc. Tipo. Fiorentina*, Firenze, 1907.

C. K. Mills and W. G. Spiller. "Symptomatology of Lesions of Lenticular Zone, with Some Discussion of the Pathology of Aphasia." Reprinted from *Journ. Nerv. and Ment. Dis.*, Aug. and Sept. 1907.

Morton Prince. "The Criminal Responsibility of Insane Persons." *Amer. Med. Assoc.*, Chicago, 1907.

Morton Prince. "The Educational Treatment of Neurasthenia and Certain Hysterical States." *Mass. Med. Soc.*

Morton Prince and Isador Coriat. "Cases illustrating the Educational Treatment of the Psycho-neuroses." *Clinic for Nerv. Dis.*, Boston.

Review of Neurology and Psychiatry

Original Articles

ON THE SPINAL CHANGES IN A CASE OF MUSCULAR DYSTROPHY.

By GORDON HOLMES, M.D.

THE muscular dystrophies are generally described as primary muscle diseases, in which the peripheral as well as the central nervous system is intact. Undoubtedly in the majority of the cases in which the nervous system has been examined, even by modern and adequate methods, no changes have been found in the lower motor neurons to which the muscular atrophy could be attributed, but in a certain number degenerative or atrophic changes have been observed in the ventral horns of the spinal cord, or in the ventral roots and peripheral nerves. The interpretation of these pathological affections of the peripheral motor neurons will need further discussion; it has already raised so much interest that a short description of another case in which it was present seems advisable. Through the kindness of Dr Beevor, under whose care the patient was when she died in the National Hospital, I have permission to give a short abstract of the clinical notes which were then taken.

E. S., a girl of eleven years of age, was admitted to hospital in July 1907. Neither of her parents nor any of their relatives were affected with similar symptoms. There were four children in the family: the eldest, a sister, was alive and well; the patient was the second member; the two younger children were boys, who were apparently healthy till about the age of two years, when each began to find difficulty in going up steps and in walking any distance;

according to the mother they were both "touched with paralysis." Both died in early childhood—the one from pneumonia, the other in a fit. The patient started to walk at the age of fourteen months, and got about naturally till eighteen months old. Then she began to fall frequently; at first she was able to rise from the ground, but later became unable to do so. She had been bedridden for a considerable time. At about the age of eight or nine years it was noticed that her arms were affected, and from that time they became rapidly weaker, till at the date of her admission to hospital she was practically helpless. The muscles supplied by the cranial nerves were unaffected, but all those of the limbs and trunk were very weak. Owing to a large amount of subcutaneous fat there was not much atrophy visible to the eye, but on palpation very little muscle tissue could be felt, and even when firmly contracted the muscles were far from firm. None of them appeared to be enlarged at this date. Those of the neck were but little affected. All the movements of the upper limbs were extremely feeble, even those of the fingers, but there was no absolute loss of power in any of the muscles except in the latissimi dorsi. The intercostals were also very weak, and breathing was chiefly diaphragmatic; the abdominal muscles were less affected. The condition of the lower limbs was very similar to that of the upper; the movements at the hip-joints were the feeblest. The limbs were very flabby, and it was noted that "there was no tone anywhere, but very marked hypotonicity in all the muscles." Sensation was unaffected. The tendon jerks of both the upper and lower limbs were absent. The reaction of all the affected muscles to the interrupted current was very much diminished, but there was no qualitative change. When in hospital the patient developed pleurisy and broncho-pneumonia, and died four weeks after admission.

A post-mortem examination was made about twenty hours after death. The muscles of the shoulder-girdle and arms were very small and soft; they were pale in colour and evidently infiltrated by fat. Of the pectorals and latissimi dorsi practically nothing could be seen but a few pale strands in the fatty tissue. The condition of the calf muscles and the glutei was very similar, the other muscles of the legs were less affected. All the peripheral nerves which were examined seemed to be somewhat smaller than they should be normally. The central nervous system was well developed, and there was no evidence of disease in it to the naked eye. The ventral

SPINAL CHANGES IN MUSCULAR DYSTROPHY 139

spinal roots were, however, remarkably small, especially those of the cervical and lumbo-sacral enlargements; they were scarcely one-third the size of the corresponding dorsal roots, and were, in addition, pinkish-grey in colour.

The microscopical examination of the affected muscles revealed in them the changes which are characteristic of the myopathies; minute description is not necessary. In the majority of those which were examined the apparent bulk of the muscle was largely occupied by fat in which muscle fibres in larger or smaller bundles were contained. Some of the fibres were apparently normal; in the deltoid and biceps the majority were so; others were very much atrophied, and contained an excess of sarcoplasmic nuclei, but their cross striation was generally well preserved; finally, in some muscles a variable number of fibres were hypertrophied. These enlarged fibres often contained central nuclei, and their cross striation was less distinct than normal. A few dividing fibres were seen. There was a variable increase of connective tissue in the affected muscles in addition to the fat. In the much wasted muscles there appeared to be a relative excess of muscle spindles; these were all intact.

No pathological changes were seen in the fore-brain or brain-stem. The white matter of the spinal cord also appeared intact when stained with the Weigert-Pal method, and no definite abnormality was detected in the grey matter, though in places the myelinated network of the ventral horns appeared somewhat rarified. The dorsal spinal roots were normal, but marked changes were at once observed in the ventral. Their small size was striking even to the naked eye, and under the microscope it was seen that not only was there a reduction in the number of the fibres, but that many of those which remained were certainly atrophied, and that the connective tissue of the roots was increased. Owing to this sclerosis the real atrophy of the roots was greater than the apparent. No accurate enumeration of the root fibres was made for comparison with the normal, but it is probable that their number was reduced to at least half.

But the most prominent change was the reduction in number and in size of the cells of the ventral horns of the cord. This was observable in all segments which were examined, but it was greatest in the cervical and lumbar enlargements. Here practically no cells of normal size could be seen, and many of those which persisted were atrophied and shrunken, and contained an amount of pigment

which was certainly excessive considering the early age at which the patient died. In the more affected cells the nuclei were small and distorted, and not distinctly visible even with Nissl's stain. In order to estimate the reduction in the number of the cells the whole of the fifth and seventh cervical, and the fifth lumbar segments were embedded in paraffin, cut in serial section, fifteen micra in thickness, and stained with thionin. The cells of the principal groups of each segment were then counted in alternate sections, and, in order to avoid enumerating each cell more than once, only those cells in which the nucleus was distinctly visible were included. By dividing the total number of cells of each group by the number of sections in which they were contained, the average number of cells per section in each group for the whole segment was obtained. The same segments of a normal cord, from a boy fifteen years of age, were treated in exactly the same way.

The results which were obtained may be given in tabular form:—

	Normal average per section.	E. S. average per section.	Percentage to which cells were reduced.
C 5 { Ventro-mesial group .	4·8	3·9	81·2
{ Ventro-lateral group .	15·8	7·6	48·1
{ Dorso-lateral group .	17·4	10·9	62·6
C 7 { Ventro-mesial group .	4	2·5	62·5
{ Ventro-lateral group .	25	6·4	25·6
{ Dorso-lateral group .	11	8·7	79
L 5 { Ventro-lateral group .	16	3·48	21·7
{ Central group .	10·2	1·57	15·3
{ Dorso-lateral group .	25	4	16

From these figures it is at once evident that there has been a considerable diminution in the number of the cells of the chief groups of the ventral horns of these segments, and that the diminution was most marked in the segment which was chosen from the lumbo-sacral enlargements. The examination of sections from other segments shows that these figures approximately represent the average loss of cells which each segment of the cervical and lumbar enlargements

at least has suffered. The enumeration of cells is such a tedious process, and requires so much time, that it was not attempted in more than these three segments. The cells of the lateral and dorsal horns and of Clarke's column were unaffected. In the portions of the ventral horns which had suffered most severely there was a slight increase of neuroglia, which was undoubtedly only secondary to the disappearance of the cells.

The atrophy and the diminution in the number of fibres of the ventral roots evidently corresponded to this loss of the ventral cornual cells and the atrophy of those which remained. Similar changes were found in many of the bundles of the nerve trunks and in the intramuscular nerves which were examined. In places there was distinct evidence of disappearance of fibres, and a variable proportion of those which remained were abnormally slender and their myelin sheaths stained badly with hæmatoxylin. In addition there was a considerable increase of the connective tissue of the endoneurium and perineurium of the nerves, but there were no signs of acute degeneration of the nerve fibres or of inflammatory or active proliferative processes in the sheaths.

There can be no doubt as to the nature of this case. The clinical symptoms, their onset at an early age and their slow evolution, as well as the familial nature of the disease, undoubtedly justified the diagnosis of primary myopathy. The histological changes in the muscles confirmed it. But as the integrity of the nervous system is generally assumed in the primary muscular atrophies, the nature and significance of these changes which were discovered in the lower motor neurons must be considered.

This is not an isolated observation ; similar changes have been described by other observers. As early as 1879 Erb and Schultze (3) recorded the disappearance of many of the larger cells of the ventral horns, atrophy of some of those which remained, and glial sclerosis in their place, in the spinal cord of a man who died at the age of fifty-eight years, with muscular atrophy which had set in two years previously. The ventral roots and peripheral nerves were unaffected. In the original communication this case was regarded as a myelopathic amyotrophy ; but later, in his well-known monograph on the muscular dystrophies, Erb (2) stated his opinion that this diagnosis was probably incorrect, and that the case was probably a muscular dystrophy of the juvenile variety.

Kahler (8) also found atrophy and pigmentation of the ventral

horn cells, but no change in the ventral roots or peripheral nerves, of a case which was accepted by Erb as a typical example of the juvenile form of muscular dystrophy.

Frohmaier (4) observed similar changes in another case of the juvenile form who died at the age of fifty-eight years, but the ventral roots were, in addition, atrophied. The symptoms probably started at about forty.

Heubner's (6) case belonged to the pseudo-hypertrophic form. The symptoms began when the patient was three, and progressed slowly till death, which occurred at twenty-one. There was a considerable diminution in the number of the larger ventral horn cells at all levels of the cord, but especially in the cervical and lumbar enlargements. There was also atrophy of many of the fibres of the peripheral nerves and of the ventral roots, with increase of connective tissue in both. The muscle changes were typical of a primary dystrophy.

Preisz (13) has also reported a case of pseudo-hypertrophic paralysis, in whom the symptoms appeared at fifteen years of age, and death took place at twenty-three. He found marked atrophy of the ventral horn cells and some increase of neuroglia in the grey matter. He does not mention if there was any numerical reduction of the cells. The fibres of the nerve trunks were small, their myelin sheaths stained badly, and the fibrous tissue of the endoneurium and perineurium was increased.

Schutz's case (17) also probably belonged to the pseudo-hypertrophic type. The disease first showed itself at the age of seven years, and death occurred at fifteen. There was a considerable reduction in the number of the ventral horn cells, and those remaining were atrophied. The peripheral nerves were described as normal.

The case which Strümpell recorded (18) was rather anomalous. The patient was a man in whom the symptoms first appeared at about the age of twenty-eight, with atrophy of the muscles of one hand; for three years the disease was limited to this arm; he died at forty. His mother had been probably similarly affected. The ventral horns were shrunken and had lost the majority of their larger cells, while those which remained were atrophied. There was also a definite loss of fibres in the ventral spinal roots and in the peripheral nerves. The author regarded his case as one of myopathy owing to the heredity, the absence of the reaction of degeneration and of

fibrillation in the affected muscles, and the character of the histological changes in the muscles, although the early distribution of the affection and the age of the patient at the time of its onset suggested that it belonged to the spinal atrophies.

In a long-standing case of the facio-scapulo-humeral variety, Sabrazès and Breugues (15) observed simple atrophy of the ventral horn cells, especially in the cervical enlargement, but without any numerical reduction. The ventral roots and peripheral nerves were normal, but probably contained an excess of small fibres.

Kollarits (9) has described atrophy, with probably reduction in number of the ventral cornual cells, especially in the cervical and lumbar enlargements, in an early case of pseudo-hypertrophic paralysis.

Rocaz and Cruchet (14) found diminution in the number of the cells, pathological changes in these persisting and glial proliferation in the ventral horns, in the same type of muscular dystrophy. The peripheral nerves were normal.

Lorenz (10) also observed a numerical diminution of the ventral horn cells in the spinal cord of a case of the juvenile form, but the cells which remained were normal.

Port (12), in another typical case of the juvenile variety, in which the onset occurred in about the nineteenth year and death took place at forty-two, also observed only a diminution in the number of the ventral horn cells.

The most recent observation is by Ingbert (7), who carefully examined the nervous system of a case of pseudo-hypertrophic palsy. He found a lessened number of cells in the lateral horns (? ventro- and dorso-lateral groups), below and including the fourth lumbar segment, and a slight increase of neuroglia in the grey matter. He measured the cross section of a number of the ventral roots and of the corresponding dorsal roots, and found, by comparing the size of the ventral roots with that of the dorsal, that the former were considerably smaller than normal. He also counted the number of fibres in the fifth lumbar ventral root (5171), and found that it contained less than half the normal number (10,366).

Thus in the cases observed by Erb and Schultze, Heubner, Schutz, Strümpell, Kollarits, Lorenz, Rocaz and Cruchet, Port, and Ingbert, as well as in my own, there was a definite diminution in the number of the ventral horn cells, while in those reported by Kahler, Frohmaier, Preisz, and Sabrazès and Breugues, these cells had only

undergone atrophy or slow degenerative changes. In the majority of these cases pathological changes were also observed in the ventral roots and peripheral nerves. In other specimens vascular or interstitial lesions were found in the cord, which were generally not limited to, and often did not affect, the grey matter, as in the case of Gowers and Lockhart Clarke (5). These were evidently only adventitial changes, and were not either directly or indirectly connected with the primary disease. Kollarits also observed pallor round the central canal and a paucity of fibres in Lissauer's zone.

It is not easy to decide at once on the origin and significance of the lesions which were found in the lower motor neurons in these fourteen cases. In my case, and in the majority of the others, there can be no doubt but that it was an acquired condition and not a developmental defect; the nature of the cell changes, the slight secondary gliosis of the ventral horns, the definite evidence of loss of fibres in the ventral roots and peripheral nerves, as well as the increase of connective tissue in the nerves, makes this certain. There are three possible explanations which may be discussed.

In the first place it is conceivable that the nervous disease was the primary and that the muscular atrophy was due to it—in other words, that the disease was of neuropathic origin. That this is possible has been put forward by Erb (2), who inclined to the view that the so-called primary muscular dystrophies are really trophoneuroses, and are dependent on either structural or functional changes in the trophic cells in the ventral horns of the cord which cannot, in the majority of the cases, be revealed by our histological methods, but of which such cases as that reported in this paper are definite evidence. Strümpell's view is somewhat similar; he assumes a defect of neurotrophic influence which first expresses itself by trophic disturbances in the portion of the neuron which is most distant from the cell—that is, in the intramuscular nerve endings. Some support was offered to this hypothesis by Sacara-Tulbure (16), who described changes in the nerve endings in a case of pseudo-hypertrophic paralysis; but, as Pick has pointed out, the histological methods which can demonstrate these structures are so imperfect that but little weight can be laid on this isolated observation.

It is generally assumed that the clinical symptoms and the mode of evolution of the muscular dystrophies separate them definitely from the myelopathic atrophies, yet there is scarcely a single symptom of the one which is not occasionally met with in the other

group, and it may be difficult to draw a sharp distinction even between the histological changes in the muscles of the two types. Despite these facts, it appears to me very improbable that the myopathies have a neuropathic basis; indeed, I cannot see how this contention can be logically sustained. In the first place, there is no reason why the muscles of certain individuals should not possess a morbid tendency to regressive changes quite as well as parts of the nervous system; and secondly, in the majority of the cases which have been examined no histological evidence of nervous change to which the muscular atrophy could be secondary has been found. I have had the opportunity of investigating the nervous system of a case of pseudo-hypertrophic paralysis which died in an advanced stage of the disease, and I failed to find any structural alterations in any portion of it, despite the use of modern technical methods. Even the case reported here, and the other thirteen cases cited in which pathological changes were found in the lower motor neurons, do not seem to me strong evidence in favour of the hypotheses of Erb and Strümpell. The histological changes in the ventral horns in my case were, it seemed to me, quite distinct from the degenerative cell changes I have found in any of the many spinal amyotrophies which I have examined, and the muscular affection exceeded them very much in both degree and extent.

In the second place, it is possible that the neural and muscular changes were the result of coincident dystrophies. I admit the possibility of this view, but I do not see any strong evidence in support of it, though undoubtedly arguments may be raised in its favour. If we assume that the nervous lesions in my case were due to a primary neural dystrophy, we find that it is connected with the most typical cases of primary myopathy, in which no nervous changes were found, by various transition types, as by Kollarits' case, in which the loss of cells was very small, and by the cases of Kahler, Frohmaier, Preisz, and Sabrazès and Breugues, in which there was atrophy but no loss of the cornual cells. If this hypothesis be accepted, it would be necessary to further subdivide the "muscular dystrophies" into the primary myopathies and the mixed neural and myopathic atrophies.

The third possibility, and that which appears to me the most probable explanation of these cases, is that the neural changes are secondary to the primary muscle disease. The view has been expressed that each lower motor neuron and the muscle fibre or

fibres in which it terminates form a biological entity, and that when the vitality of any part of this unit is seriously disturbed, the rest will react to the lesion and may undergo fatal regressive changes. It is unnecessary to refer to the atrophy of the muscle fibres which results from a neural lesion, but the evidence of the converse process is not so evident. It is true that reactionary changes can be generally found in some part of the neuron when the muscles in which it terminates have been removed by amputation, or destroyed by a tumour, but then part of the neuron itself is evidently injured. I know not of any evidence of neural degeneration secondary to disease entirely limited to muscle tissue, but it seems very probable that when muscle fibres undergo complete atrophy and disappear, the functional, and probably secondary thereto the nutritional, equilibrium of the neurons which terminate in these fibres must be disturbed. And further, in this case, as in the example of muscular dystrophy which I have described, the terminal branches of the axiscylinder, left naked by the disappearance of the muscle fibres, will be probably injured and possibly destroyed by the connective tissue which proliferates secondary to the muscle disease. This is the explanation which I am inclined to adopt of the pathological changes which have been found in the peripheral nerves and the ventral horn cells in a considerable number of cases which have been regarded as primary muscle diseases. At the same time I admit the possibility of the hypothesis already put forward, according to which the muscular and neural changes are due to a coincident tendency to muscular and neuronie degeneration.

It remains to consider why these nervous changes, if they are secondary to the muscle disease, occur in some cases of primary myopathy and are absent in others. Evidently the clinical variety of the disease is unimportant, as the cases of Heubner, Preisz, Schutz, Kollarits, Rocaz and Cruchet, and Ingbert belonged to the pseudo-hypertrophic form, while those of Erb and Schultze, Kahler, Frohmaier, Strümpell, Lorenz, Port, and probably my own, were of the juvenile type, and that recorded by Sabrazès and Breugues was an example of the facio-scapulo-humeral variety.

The age at the onset of the symptoms, it might be thought, would have some influence on the occurrence of these secondary nervous changes, as the degeneration of muscle fibres in early childhood could possibly arrest or inhibit the development of the neurons concerned in their nutrition; but an analysis of these fourteen cases

shows that in many instances, as in those of Erb and Schultze, Frohmaier, Strümpell, and Port, the muscle disease set in relatively late in life. The long duration of the disease does not seem to be an essential factor. The integrity of the nervous system in some cases of primary muscular atrophy, and the atrophy or degeneration of some of the lower motor neurons in other cases, is parallel to the fact that sometimes all the ventral horn motor cells disappear after amputation of the segment of the limb which they supply, while in other cases these cells may be practically unaffected.

Reference may be made here to a case which has been reported by Baudouin (1) as myatonia congenita, owing to the resemblance the pathological changes which were discovered in it bore to those in my case. The patient was evidently affected from birth, and died at the age of four months. The clinical symptoms were regarded as typical of myatonia congenita, as this disease was originally described by Oppenheim. The functions of the cranial nerves were unaffected, excepting slight strabismus which may have been only an incidental symptom, but from birth the limbs, neck, and trunk were powerless and flabby. There was practically no power of movement in the proximal joints of the limbs, but slight of the toes and fingers. The muscular hypotonia was very marked, and the Faradic excitability of the muscles was very much reduced. Sensation was unaffected; the tendon jerks were absent. On microscopical examination it was found that the muscles were largely replaced by fat, and contained a considerable excess of fibrous tissue; the majority of the fibres which remained were atrophied and contained a large number of sarcoplasmic nuclei, but others were enormously hypertrophied and were penetrated by nuclei. Some fibres were dividing. There was a relative excess of muscle spindles. In fact, the muscular changes were identical, as the illustrations show, with those which are regarded as pathognomonic of the primary muscular dystrophies; the author himself remarks, "*les lésions de la régression musculaire, telles qu'on peut les voir dans les myopathies.*" But there was in addition a considerable diminution in the volume and possibly in the number of the ventral horn cells, a reduction of the ventral roots to less than half their normal size, and in these and the peripheral nerves there were atrophied fibres and secondary fibrosis. The similarity of the histological changes in this case to those in the muscular and nervous systems of my case needs no further mention; in fact, the morbid anatomy of

Baudouin's myatonia congenita was identical with that of a considerable number of undoubted cases of muscular dystrophy. In origin, too, they were probably similar, for, as Baudouin points out, the abnormal condition of the muscles could not be regarded as due merely to an arrest of development, it must have been an acquired condition. Owing to its intensity it could scarcely have been secondary to the simple atrophy of the ventral horn cells, and its nature too was quite distinct from that of the neuropathic atrophies. It must be concluded, then, that the disease of the muscles was an acquired regressive affection, dependent probably on an intrinsic developmental anomaly. Let it be remembered that till birth at least the development of the muscles is independent of the nervous system, as has been demonstrated by the presence of normal muscles in cases of amyelia. From the pathological point of view, therefore, this case can be regarded as a primary muscular dystrophy, exceptional only in the fact that the disease commenced during intra-uterine life. The clinical symptoms of the case also resembled those of the muscular dystrophies so closely that it seems unnecessary to place it in a separate class. There was not only atonia, but practically complete paralysis of the muscles, and the muscles of the basis of the limbs were more affected than those of the distal segments. There was quantitative, but no qualitative, change in the electrical reactions, the deep reflexes were absent, and sensation was not affected. From the clinical point of view, too, the case was therefore unlike the myopathies only by the fact that the disease started so early in life. Experience has already shown that it is impossible to place a limit in the one direction to the age at which the symptoms of muscular dystrophy may set in—cases have been recorded which commenced after sixty years of age—and it seems equally impossible to set up a limit in the other.

It may be that other cases have been included in myatonia congenita which have been only instances of myopathy of exceptionally early or congenital origin. Indeed, cases have been reported under this title in which the origin of the disease was undoubtedly post-natal. It is not my intention to deny the existence of that form of disease which Oppenheim has described as a pathological entity, but there seems to be a danger of including cases of different nature within it. It should be recognised that muscular atonia is a prominent symptom of the myopathies, unless the extensibility of the muscles is restricted by the formation of fibrous tissue within

them. If, on the other hand, contractures developed in some muscles in a case of myatonia congenita, owing to the atonia of their antagonists or to the deposition of fibrous tissue within them, as would probably have occurred in Baudouin's case if the child had lived, so that the mobility of the joints was lessened and not excessive, the case would not be, it may be assumed, immediately rejected from Oppenheim's group if the other symptoms conformed to the type.

The most important and definite distinction between myatonia congenita and the muscular dystrophies seems to be that in the former the symptoms are not progressive and that amelioration may occur. Even if these cases, or any of them, were congenital muscular dystrophies, this feature would not be surprising in view of the difference of the nutritional conditions of the individual before and after birth, and the change in the mutual relationship of the muscular and nervous systems in intra- and extra-uterine life.

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THE MYASTHENIC REACTION EXPERIMENTALLY PRODUCED IN THE FROG.

By J. A. GUNN, M.D., B.Sc., M.A.,

Assistant in the Materia Medica Department, Edinburgh University.

IN the course of an investigation of the pharmacological action of yohimbine, I found that there is produced in frogs, by the administration of large doses of this alkaloid, a condition of rapid muscular exhaustion, the symptoms of which present a close similarity to those which obtain in the disease in man, myasthenia gravis. Further inquiry showed that this resemblance is not a mere superficial one, but that the electrical reactions of the nerves and muscles of the yohimbinised frog are identical with those which have been generally found in myasthenia gravis, and which are designated by the term "myasthenic reaction," and associated with the name of Jolly, who first described them.

Seeing that post-mortem examinations of cases of myasthenia gravis have as yet failed to demonstrate lesions which can adequately explain the phenomena of this disease, the experimental production of a similar condition is not merely interesting, but may also be of importance in so far as pharmacological experiment affords wider facilities for the exact determination of the site of this muscular exhaustion. Though identity of result does not of necessity mean identity of cause, still the fact that the artificially produced effects of a toxic agent like yohimbine are identical with the effects produced by a presumable toxin operating in myasthenia gravis, indicates certainly the possibility, even the probability, that the nature of the action of the causes producing these effects is the same in the two cases.

In order to institute this comparison it is convenient, first, to review briefly the condition known as myasthenia gravis; secondly, to describe the similar condition produced in the frog by yohimbine; and, finally, to endeavour to locate as far as possible the site of the action of yohimbine.

In myasthenia gravis "there is weakness, sometimes amounting to complete paralysis, of some or all of the voluntary muscles—*e.g.* the first thing in the morning they may respond normally to

the will, but they become rapidly exhausted after voluntary contractions, regaining their power again after rest. The entire system of voluntary muscles may be affected, but those muscles are most apt to be implicated which normally act most constantly, such as the cervical muscles and the extrinsic muscles of the eyeballs. The bulbar muscles are very generally involved. Hence the term 'asthenic bulbar paralysis.' A characteristic feature of the disease is its tendency to fluctuate in severity from day to day, or from week to week, or even to disappear for months, to reappear" (1).

Jolly found in a myasthenic patient that if a striped muscle be subjected to faradic stimulation, either directly or by way of its nerve, after a short time the muscle manifests fatigue of the same nature as ensues after voluntary stimulation of the muscles. If faradic stimulation be alternately applied for a few seconds and discontinued for a few seconds, the resulting tetanus of the muscle becomes less and less complete with each successive stimulation, until a stage is soon reached in which only at the entrance of the stimulation is there elicited a transient contraction (like the contraction from making a constant current), while during the continuance of faradisation the muscle response becomes quite weak or eventually disappears. By either increasing the strength, or by applying the same strength, of current after a minute's rest, these phenomena are repeated. If, instead of alternately applying and discontinuing the stimulations for periods of a few seconds, the muscle be faradised continuously for a minute, a similar diminution in the muscular response results even to its ultimate disappearance (2).

This fatigue effect is called the "myasthenic reaction." It "clearly demonstrates an extraordinary inaptitude for prolonged muscular activity. The same condition is reflected in the volitional actions of such patients" (3).

How far are the effects produced in frogs by yohimbine comparable with the conditions in myasthenia gravis?

Symptoms of Muscular Exhaustion in Frogs after Subcutaneous Injection of Yohimbine.—I have found that the minimum lethal dose of yohimbine lactate for male frogs (*Rana temporaria*) is 0.05 gm. per kilo of frog weight. After similar administration of toxic, but sublethal, doses of yohimbine lactate (*e.g.* 0.04 to 0.048 gm. per kilo) there occur in frogs symptoms of

paralysis, due, firstly, to an action on the central nervous system ; and, secondly, to an action on the peripheral neuro-muscular mechanism, and respectively early and late in their onset.

The symptoms of the former action appear in about two hours, and may be dismissed briefly, as they are not primarily concerned in the myasthenic reaction. They consist of paralysis of the functions of the mid-brain, cerebellum and medulla, witnessed by inco-ordination of movement, by loss of the power of jumping and of recovering its position when the animal is laid on its back, and by arrest of the respiration. At this period the voluntary muscles and their motor nerves react normally to electrical stimulation, and there is no evidence of rapid muscular exhaustion on the part of the frog. For example, when pinched he responds by vigorous and sustained kicking, though he is unable to jump away. If a hind limb be drawn down several times, it is each time rapidly pulled up again to its usual flexed position.

Later, however, in about four or five hours after injection, other symptoms appear. The response to pinching, though as prompt as heretofore, and still elicited by slight stimulation, is ill-sustained. If a hind limb be now drawn down once it is rapidly pulled up, but if drawn down a second time it remains extended. A few movements, therefore, suffice to bring on fatigue. The functions of the central nervous system are recovering so that he may be able to give one vigorous jump, but after this effort he is incapable of further movement. After a very short rest this power is regained, only to disappear after renewed voluntary movement. Substituting "intoxication" for "disease," the description applied to myasthenia gravis is equally applicable to the symptoms in the frog during this stage of yohimbine poisoning—"the speedy production of muscular exhaustion constitutes the most striking feature of the disease. Persistence in a movement causes it to become gradually weaker, and ultimately impossible, until after a short rest, when it can again be repeated" (4).

Generally by the end of twenty-four hours after injection of yohimbine in the doses mentioned the frog recovers from these symptoms of unduly rapid onset of fatigue.

One symptom is interesting, though it may be a coincidence and not significant—namely, rise of the lower eyelids of the frog.

This symptom is a herald of onset of the muscular exhaustion which later appears in the limb muscles. The lower eyelid of the frog is depressed by muscular effort, and rises when this effort ceases. Its rise, therefore, corresponds to ptosis in man. In myasthenia gravis, ptosis is very frequently also one of the earliest symptoms. It is at least an indication that myasthenia gravis may be due to a toxin having a similar action to yohimbine, the more so as there is to hand an example in cobra poisoning of a toxin which produces ptosis in man, and in the frog elevation of the lower eyelids (5).

There is thus manifestly a singular correspondence in the clinical picture of myasthenia gravis and yohimbine poisoning. The question now arises as to how far this symptomatic resemblance is intimate in nature, and how is the muscular fatigue in the latter condition to be explained.

It is convenient first to consider :

Faradic Stimulation of the Motor Nerve.—In order to investigate this a frog was killed during the fatigue stage of yohimbine poisoning, and a gastrocnemius muscle with its sciatic nerve isolated immediately, and kept moistened with Ringer's solution. A du Bois Reymond coil with Neef's hammer was used to stimulate the nerve, which was laid on platinum electrodes. The femoral attachment of the gastrocnemius was fixed, and the tendo-Achillis connected by a thread to a lever, which recorded the muscular response on a slowly-revolving smoked drum. The results of such an experiment are shown on Figs. A and B.

Faradic stimulation of the sciatic nerve with the secondary coil at 240 mm. evoked no muscular contraction (A 1).

With the secondary coil at 160 mm. a full tetanic contraction of the muscle was obtained. Though, however, the nerve was stimulated for twenty seconds, tetanus of the muscle lasted only ten seconds. During the remaining ten seconds the muscle gradually relaxed, and eventually responded by only the feeblest contractions (A 2).

After an interval of eight seconds the nerve was again stimulated for over ten seconds, and an incomplete and evanescent tetanus of the muscle was elicited. During the remainder of the stimulation no effect was produced on the muscle (A 3).

With subsequent faradisations, separated by a shorter interval

of time, there resulted a contraction only at the moment of entrance of the current, and this contraction became feebler and feebler (A 4, 5, and 6), and eventually disappeared (A 7).

After a short interval of rest the muscle again responded, the vigour of this response varying with the length of rest interval elapsing between the stimulations (A 8, 9, and 10).

After a rest of three minutes there was almost complete restoration of the nerve-muscle to its previous condition (B 1), but subsequent repeated stimulations separated by a short interval repeat the effects seen in Fig. A.

If the nerve of a normal gastrocnemius-sciatic preparation be stimulated under the same conditions of experiment, complete tetanus of the muscle lasts for many minutes.

Direct Faradic Stimulation of the Muscle (Fig. C).—As before, a frog was killed during the fatigue stage of yohimbine poisoning, and a gastrocnemius muscle isolated immediately. The muscle was stimulated directly. Fig. C 1 shows the height of a single muscle twitch under these conditions. The muscle was then directly faradised continuously for seventy seconds. As the stimulation went on, the muscular response, beginning with complete tetanus, degenerates into irregular contractions, which finally disappear (C 2).

Subsequent faradic stimulations, each lasting a few seconds, and separated by an interval of a few seconds, induced a contraction only at the beginning of stimulation. These resembled a single muscle twitch (C 3 and 4). These contractions became less and less on repetition of the stimulations, and finally disappeared (C 5, 6, and 7).

With successively longer periods of rest, the stimulations produce a more and more effective tetanus (C 8, 9, and 10), to become less effective when the interval of rest is made shorter (C 11, 12, and 13).

There is therefore on direct faradisation an unduly rapid onset of exhaustion of the yohimbinised muscle, as compared with the behaviour of a normal muscle under the same conditions.

The electrical reactions of the yohimbinised muscles are therefore identical with those which have been described by Jolly in myasthenia gravis.

Faradic versus Galvanic Stimulation of the Muscles (Fig. D).—In myasthenia gravis some importance has been ascribed to the

fact that the muscles are much less readily exhausted by galvanic than by faradic stimulation. In order to determine whether this phenomenon is also a feature of the yohimbine-poisoned muscle, arrangements were made whereby, with the help of two commutators and a mercury dip key, the muscle could either be faradised or stimulated by the make or break of a galvanic current, only such time elapsing between these stimulations as would allow of reversing the commutators.

The results of such an experiment are shown in Fig. D, where once more a yohimbinised muscle preparation was used. Fig. D 1 shows three contractions obtained by making the constant current.

The muscle was then continuously faradised for twenty seconds. At first a good tetanic contraction of the muscle was obtained, but as the stimulation went on the muscle soon relaxed, and eventually ceased to respond (D 2).

A few seconds later the muscle was stimulated by making the constant current for fifty seconds at the rate of about once a second. The resulting contractions were almost as good as before the muscle was exhausted by tetanus (D 3).

The muscle was then exhausted by repeated tetanisations separated by increasingly short intervals of time. As has been similarly shown in Fig. C, the muscle contracted only at the moment of commencing stimulation. After very short intervals of rest, the muscle refused to contract. A few seconds later, however, the muscle still responded very well to the makes of the galvanic current (D 7).

In regard to this test, too, the muscles react like the muscles of a myasthenic patient.

Cause of the Myasthenic Reaction Produced by Yohimbine.—There is, I think, nothing in these results to decide absolutely in favour of the abnormally rapid onset of fatigue in this condition being due to an exhaustion of the nerve alone or of the muscle alone. As compared with a normal muscle, a yohimbinised muscle, whether stimulated directly or by way of its motor nerve, is incapable of sustained tetanus. Direct stimulation of the muscle does not, however, exclude stimulation of the intramuscular nerves.

Certain other ascertained facts of the action of yohimbine aid in a solution of this problem. Thus Tait and I have found in regard to motor nerve that the myasthenic reaction can be

produced by the direct application of a two per cent. solution of yohimbine lactate to the nerve trunk. We have found that by subjecting a small portion of the middle of the sciatic nerve to such a solution, the muscular response which results from faradic stimulation of the proximal end of the nerve varies with the interval of time between the stimulations. At first the muscle responds with tetanus. With a short interval of rest between successive faradisations a contraction occurs only at the beginning of stimulation, or there may be even no muscular response. Faradisation of the same part of the nerve again produces tetanus, provided a sufficient interval of rest is allowed. In this case there can be no question of a direct effect of yohimbine either on the nerve ends or on the muscle, for faradisation of the part of the nerve distal to the yohimbinised portion elicits a normal tetanus which shows no such impairment on continuous or repeated stimulation (6).

It is therefore practically certain that the more dilute solution of yohimbine which finds itself in the blood of the frog after the subcutaneous injection of this drug will produce, either by affecting the nerve trunks or more probably the finer nerve fibres in the muscle, such effects as can in great measure at least explain the myasthenic reaction. It is also apparent that this action may be entirely a primary one, secondary to an affection neither of the muscle nor of its nerve ends, nor of the motor cell from which the nerve arises.

As to whether yohimbine also affects the nerve ends, it seems difficult to decide. The usual tests by which the curara action can be localised to the nerve ends fail on account of the powerful action which yohimbine exerts on the nerve trunks.

There is as yet no definite consensus of opinion regarding the site of the exhaustion in myasthenia gravis. For example, Jolly suggests a dystrophy of the voluntary muscles, Myers a toxic action on the nerve ends in the muscle, and Campbell and Bramwell a toxic action on the axons. Others have supposed, probably with less likelihood, an affection of the central nervous system.

With regard to pharmacological evidence, Jolly considers that certain experiments performed on the frog with protoveratrine by Watts-Eden support his hypothesis of a muscular explanation.

Watts-Eden (7) describes symptoms of motor exhaustion occurring in the frog after injections of protoveratrine similar to those produced by yohimbine. He found, among other things, that a muscle treated with a saline solution containing protoveratrine is rapidly fatigued by a few single induction shocks, whereas if the nerve be soaked in a similar solution for a prolonged period there is no such loss of excitability. Jolly took this as supporting his views of a muscular explanation.

I have found that a muscle treated with a saline solution containing yohimbine reacts in practically the same way, but unfortunately this method of experiment does not get rid of the intramuscular nerve fibrils, and in point of fact Waller (8), by investigating with the galvanometer the electrical response to stimulation, found that protoveratrine exerts a distinct effect on the motor nerves. He found that protoveratrinised nerve, when subjected to a series of tetanising stimulations, "exhibits a well-marked negative sign of action during excitation, but little or no positive sign of reaction after excitation. And in correspondence with this failure of positive reaction, the succession of action-effects exhibits rapidly progressing exhaustion."

As Myers points out, this finding of Waller's, together with Watts-Eden's experiments showing that protoveratrine has a paralysing action on sensory nerves, would seem to show that this substance has a general action on nerve fibres.

Yohimbine also paralyses sensory nerves, and its action on motor nerves is remarkably like that of protoveratrine; and I have further shown that this action in the case of yohimbine is adequate to explain the myasthenic reaction. It is suggestive that two alkaloidal substances which have been noted as producing effects similar to myasthenia gravis should be found on analysis to have actions on nerve so similar to one another.

The balance of pharmacological evidence, so far as such evidence can be translated to the interpretation of pathological conditions in man, seems therefore to rest in favour of the myasthenic reaction in myasthenia gravis being due, at any rate in part, to a toxic action on the motor nerves.

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DESCRIPTION OF FIGURES.

- A. Faradic stimulation of sciatic nerve. The signal shows the time during which the nerve was faradised.
 - B. Continuation of A, three minutes later.
 - C. Faradic stimulation of gastrocnemius muscle.
 - D. Faradic versus galvanic stimulation of the gastrocnemius muscle.
- The signal (2, 4, 5, and 6) shows the time during which the muscle was faradised. The single contractions without signal (1, 3, and 7) are from makes of a constant current.

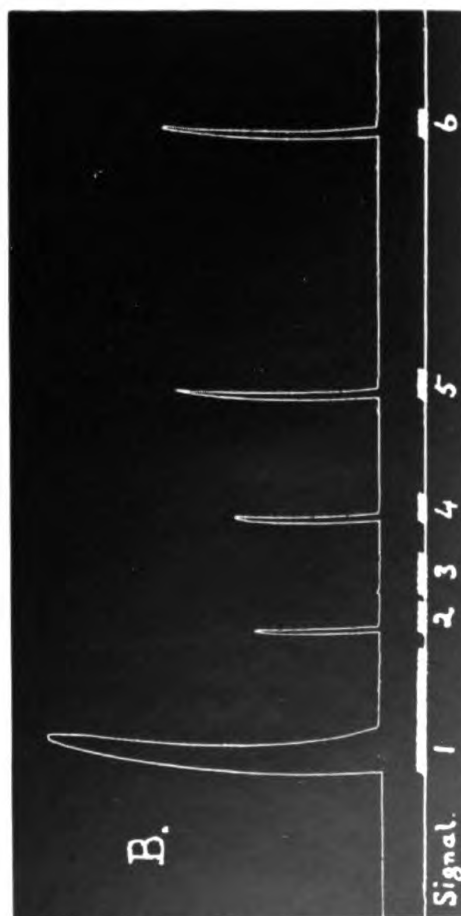
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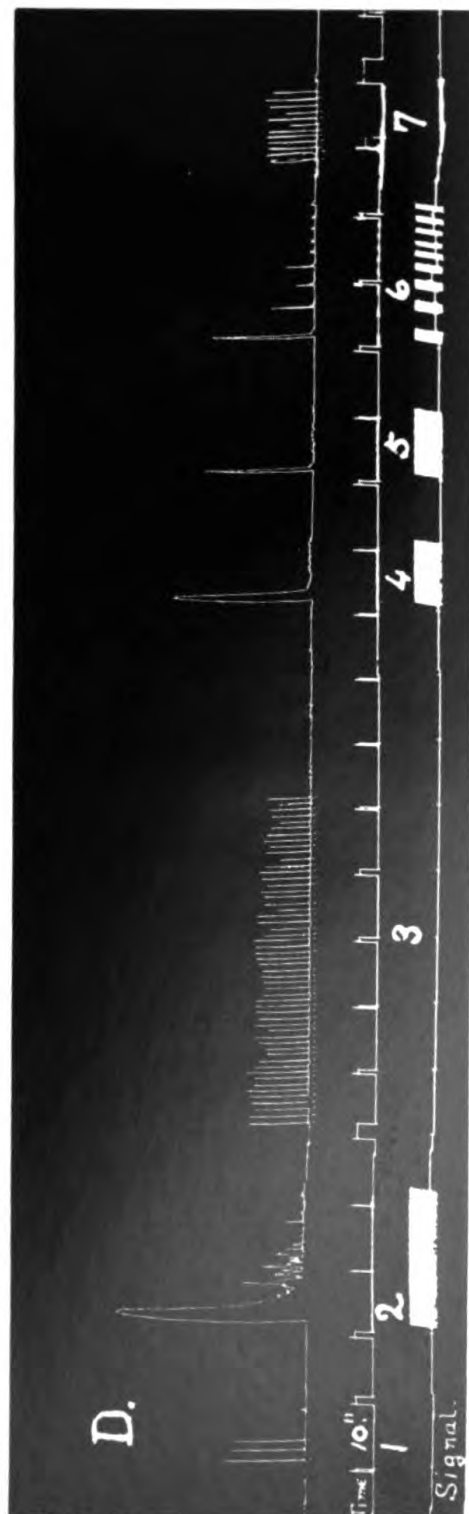
ANATOMY.

THE ANATOMY AND DEVELOPMENT OF THE INDUSEUM

- (112) **GRISEUM CORPORIS CALLOSI.** (Zur Anatomie und Entwicklungsgeschichte des Indusium griseum corporis callosi.)
E. ZUCKERANDL, *Arb. a. d. Neurol. Instit. Wien*, Bd. 15, S. 17, 1907.

THE grey matter known as the indusium griseum which covers the dorsal surface of the corpus callosum consists of two different parts of different origin; the lateral symmetrical portions undoubtedly belong to the gyrus supracallosus or the dorsalward prolongation of the cornu ammonis, while the median portion consists of fibres some of which penetrate the corpus callosum as the fibræ perforantes of Kölliker. This paper is devoted to the investigation of the origin and course of these fibres of the median or intermediate portion of the indusium griseum. It was found that this portion is probably present in all mammals, though in some species it is so small that its demonstration is difficult; in fact, an examination of the reptilian brains shows that it is one of the fundamental cerebral structures.





Its fibres arise in part from the gyrus supracallosus. It develops from the septum, with a part probably from the lateral walls of the hemispheres. It is at first a bilateral and symmetrical structure, but during its development its two parts fuse together.

GORDON HOLMES.

THE COMPARATIVE ANATOMY OF THE NUCLEUS RUBER

(113) **TEGMENTI.** (*Zur vergleichenden Anatomie des Nucleus ruber tegmenti.*) R. HATSCHKE, *Arch. a. d. Neurol. Instit. Wien*, Bd. 15, S. 89, 1907.

IN this paper the form, constitution and chief connections of the nucleus ruber in the different classes of mammals are described. Its fundamental nature is most easily studied in one of the lower monkeys, in which its different parts are well developed. Here it is easily seen that the nucleus consists of two distinct parts: the one, which contains only large cells, occupies the dorsal and caudal portion, and is named by the author nucleus magnicellulatus; the other is composed of much smaller cells, and forms the frontal and ventral portion of the nucleus—nucleus parvicellulatus. In the lower mammals the former is much the larger, in the carnivora it forms quite two-thirds of the whole nucleus, but it becomes relatively much smaller in the higher apes, and in man it is only a rudiment represented at most by a group of eight to ten cells in a section, at the dorsal and caudal border of the nucleus ruber proper. As the nucleus magnicellulatus diminishes in size the nucleus parvicellulatus increases, and in the anthropoid apes and in man forms practically the whole of that which is generally described as the nucleus ruber.

Hatschek has not been able to furnish any new evidence on the efferent connections of these two parts of the nucleus ruber, but he cites the work of Kohnstamm and Preisig, who found chromatolytic changes only in the cells of the nucleus magnicellulatus after section of the tractus rubro-spinalis, and who have thus proved that these cells send their axiscylinders spinalwards. On the other hand, Preisig has demonstrated changes in the small-celled nucleus as the result of a section between the nucleus ruber and the optic thalamus, thus proving that these cells send their axiscylinders forwards. The latter nucleus seems to be an important co-ordination centre intercalated between the cerebellum and the fore-brain.

On examining the cerebellum through the mammalian series, Hatschek has also found that the nucleus dentatus increases in size, step by step with the nucleus ruber parvicellulatus, while the development of the medial cerebellar nuclei, the embolus and globosus, which are relatively much larger in the lower mammals, corresponds

with that of the large-celled portion of the nucleus ruber. In the lower mammals the superior cerebellar peduncle crosses the middle line in two distinct decussations, a dorsal and a ventral. In those species in which the dorsal decussation predominates, the nucleus magnicellulatus is much larger than the nucleus parvicellulatus, while in the higher mammals the majority of the fibres, and in man practically all, cross in the ventral decussation. It therefore seems that the fibres of the superior peduncle which arise from the embolus and nucleus globosus cross the raphe of the mid-brain in the dorsal decussation and are in intimate connection with the nucleus ruber magnicellulatus, and that those from the nucleus dentatus pass through the ventral crossing and terminate in the nucleus parvicellulatus.

GORDON HOLMES,

THE FIBRILS AND THE FIBRILLOGENOUS SUBSTANCE IN
 (114) **THE GANGLION CELLS OF VERTEBRATES.** (*Le fibrille e la sostanza fibrillogena nelle cellule ganglionari dei vertebrati.*) O. FRAGNITO, *Annali di Neurol.*, Anno xxv., Fasc. 3, 1907.

WE have in this paper a further contribution to the question of the development of the fibrils in the nerve cells of the cord of the chick, and a reply to the criticisms of Cajal on the author's previous work.

In this research the author has employed the Vth method of Donaggio for staining nerve fibrils, with slight modifications. With this stain the chromatic substance is stained a clear blue, while the fibrils have a reddish-violet tint, in the cells of embryos as well as of adults. At a period of development, when the fibrils are wanting or have just begun to appear, it is possible to recognise constantly in the cells two substances—one, which has assumed a blue colour and is distributed throughout the cell; and a second, which has a reddish-violet tint and occupies the central portion of the cell, lying usually beside the nucleus. This latter he calls the "fibrillogenous substance." With the aid of a very interesting series of illustrations, the differentiation of the fibrils from this fibrillogenous substance and the connection of the fibrils with the processes of the cell are clearly demonstrated.

It is evident, then, that the conducting element of the adult nerve cell "makes its first appearance in the protoplasm of the ganglion cell, not in the form of neurofibrils, but of an undifferentiated substance, from which they develop later." This undifferentiated substance can be seen in most of the cells of the cord of a chick on the tenth and eleventh days of development,

and the differentiation of the fibrils can be followed in subsequent stages of development.

In the earlier stages of development Fragnito has found that the fibrillogenous mass has a fairly clear outline, and resembles closely in size and shape the nucleus of the nerve cell. By examining the cord of a chick at the ninth day of development he has seen that the nerve cells have two nuclei, each of which is furnished with a nucleolus and a dense chromatic reticulum. Between this condition and the appearance of the fibrillogenous mass a series of intermediate stages have been observed, in which the second nucleus loses its sharp outline, and the fibrillogenous substance is seen to be derived from its nucleolus.

The author then refers to the fact that many observers have recognised that there are substances in the cytoplasm of the nerve cell which are of nuclear origin, and it has been suggested by them that these substances have been secreted by the nucleus, and have passed out from it, in the form of globules or as a fluid, to constitute the cytoplasm. Fragnito, however, is of the opinion that the chromophile substance of the nerve cell is derived from the nuclear substance of the secondary neuroblast.

From the more recent investigations it seems probable that, at least in the cord of the chick, the adult nerve cell is derived from two neuroblasts, one of which develops into the adult nucleus, and the other undergoes certain changes and gives rise to the cytoplasm and the fibrils.

R. G. Rows.

ON THE COURSE OF THE AFFERENT PORTION OF A NUMBER OF REFLEX ARCS, ESPECIALLY OF THE LOWER SPINAL REFLEXES. (Ueber den radiculären Verlauf des centripetalen Teiles einer Anzahl von Reflexbogen, besonders von Reflexen des untersten Rückenmarksabschnittes.) G. BIKELES und W. FROMOWICZ, *Arch. a. d. Neurol. Instit. Wien.*, Bd. 15, S. 52, 1907.

A LARGE number of skin, sphincter and tendon reflexes can be obtained in dogs in which the spinal cord had been previously transected in the lower thoracic or upper lumbar region; some of these reflexes can be constantly obtained in every animal, others are present in only some specimens. The dorsal roots through which the afferent portions of these reflexes pass were determined by exposing the cauda equina and cutting through a series of dorsal roots in succession, noting when the reflex disappears.

Eighteen different reflexes were investigated in this way, but as long descriptions of the form of each reflex and of the mode of

exciting it would be necessary, only a few of the more important can be referred to here.

All the sacral and the first coccygeal roots carry afferent fibres concerned in the true anal reflex; reflex depression of the tail on stroking the skin of the perineum is no longer obtained when the second sacral to the second coccygeal roots are cut; the seventh lumbar root contains the afferent fibres for the Achilles-tendon reflex, and reflex flexion of the toes is lost when the sixth lumbar root and the roots below it are cut.

GORDON HOLMES.

THE ORIGIN OF THE SUPERIOR FACIAL IN MAN. (L'origine (116) du facial supérieur chez l'homme.) C. PARHON and J. MINEA, *Presse Méd.*, No. 66, 1907, p. 521.

THE authors have been fortunate enough to have had an opportunity of examining a case of paralysis limited to the upper facial muscles—namely, the frontalis, the corrugator supercilii and the orbicularis palpebrarum. The paralysis was due to the involvement of the upper branches of the nerve by a circumscribed epithelioma. They found an atrophy of the dorsal group of nerve cells in the common nucleus of the facial. Further examination showed that the nuclei of the 3rd, 4th, and 6th nerves were quite intact, as also was the group of cells in the neighbourhood of the 3rd and 4th nuclei, which occupies a depression on the posterior longitudinal fasciculus, and which is regarded by Giannelli as the nucleus for the upper facial muscles. They attribute the frequent escape of the superior facial muscles in certain atrophies of nuclear origin to the fact that the nucleus of the upper branch of the facial nerve forms a perfectly circumscribed group quite independent of the rest of the facial nucleus.

ALEXANDER BRUCE.

PHYSIOLOGY.

IS THE UPRIGHT POSITION MAINTAINED BY SENSATIONS
(117) **FROM THE JOINTS?** (Wird der Stehende durch das Lagegefühl der Glieder (durch die Nachricht über Gelenkstellungen) vor dem Fallen bewahrt?) S. ERBEN, *Arch. a. d. Neurol. Instit. a. d. Wien. Univ.*, Bd. xvi., T. 2, 1907, S. 23.

It is shown that a person in the erect attitude balances his body and keeps himself from falling not by any information derived from the joints of the lower limbs, but by sensory impressions conveyed from the sole of the foot. Increased pressure on the heel, *e.g.*, tells the person that the vertical line from the centre of gravity of the

body is about to fall outside his base of support behind. If the pressure increases towards the ball of the foot, he knows that he is going to fall forward if the body is not thrown backward to prevent it.

In locomotor ataxia the patient no longer receives these sensations from the sole of the foot, and if his eyes be closed so that he cannot see the relative positions of objects around him, he has no guide at all and so cannot maintain his balance.

SUTHERLAND SIMPSON.

FURTHER DATA REGARDING THE CONDITION OF THE
(118) **VASO-MOTOR NEURONS IN "SHOCK."** W. T. PORTER and
W. C. QUINBY, *Amer. Journ. Physiol.*, Vol. xx., No. 4, p. 500.

It is generally held that in "shock" the vasomotor cells are depressed, exhausted or inhibited by the excessive stimulation of afferent nerves, and that the fall in blood pressure and the accompanying symptoms are due to this depression.

In this paper the authors give the results of experiments which appear to disprove this theory. They stimulated the depressor nerve in the rabbit and noted the effect on the blood pressure before and during shock (produced by violent peripheral excitation), and they found that the "percentile fall" in the blood pressure before and during shock was about the same. By "percentile fall" they mean the difference between the blood pressure before and during stimulation of the depressor calculated to percentages. If the vasomotor centre is exhausted in shock, any additional afferent stimulation ought to have little effect on the blood pressure, nevertheless they found that the relative effect is the same.

SUTHERLAND SIMPSON.

SOME COMPARISONS BETWEEN REFLEX INHIBITION AND
(119) **REFLEX EXCITATION.** C. S. SHERRINGTON, *Quar. Journ. Exper. Physiol.*, Vol. i., No. 1, p. 67.

THE author has succeeded in demonstrating the fact that by gradually increasing the strength of the external stimulus the reflex effect is proportionately increased. It used to be believed that the "all or none" phenomenon held for spinal reflex arcs as well as for cardiac muscle.

The reflex studied was the flexion-reflex in the hind limb of the decerebrate dog. This is a reflex of simultaneous double sign (\pm), contraction of the muscles which produce flexion of the knee-joint occurring at the same moment as those which cause extension of that joint. A single muscle of each of the opposing groups was

selected to act upon the joint—the semi-tendinosus (flexor) and vaso-crureus (extensor). The exciting stimulus was a single induction shock obtained from a Kronecker inductorium, and it was applied to the same afferent nerve in all the experiments, viz., the musculo-cutaneous branch of the peroneal, about 4 cm. below the knee.

The reflexes obtained from the isolated semi-tendinosus show that by increasing the strength of the stimulus both the amplitude and the duration of the contractions increase. The same holds on the inhibitory (relaxation) side of this reflex,—with increase in the strength of the stimulus the amplitude and speed of the reflex relaxation increase.

On the excitatory side of the reflex there is no superposition unless the shocks succeed each other at intervals shorter than .15 sec. With this the inhibitory part of the reflex stands in apparent contrast, because superposition (successive elongation) occurs when the stimuli are applied at the rate of only four or five per second. Thus stimuli which, taken singly, produce almost no perceptible relaxation of the vaso-crureus, when repeated even at the above comparatively slow rate lead to extensive relaxation by summation.

SUTHERLAND SIMPSON.

**ON PROTAGON : ITS CHEMICAL COMPOSITION AND PHYSICAL
(120) CONSTANTS, ITS BEHAVIOUR TOWARDS ALCOHOL
AND ITS INDIVIDUALITY.** R. A. WILSON and W. CRAMER,
Quar. Journ. Exper. Physiol., Vol. i., No. 1, p. 97.

WITHIN recent years it has been asserted that the substance prepared from brain tissue known as protagon is not one but a mixture of several substances. The authors, from the examination of many samples prepared by different methods, conclude that it has a definite chemical composition and constant physical properties—in other words, that it is a definite chemical substance and not a mixture.

SUTHERLAND SIMPSON.

**ON THE TIME TAKEN IN TRANSMISSION OF REFLEX
(121) IMPULSES IN THE SPINAL CORD OF THE FROG.**
FLORENCE BUCHANAN, *Quar. Journ. Exper. Physiol.*, Vol. i.,
No. 1, p. 1.

NON-POLARISABLE electrodes connected with a capillary electrometer arranged for obtaining photographic records were led off from one gastrocnemius muscle of a decerebrate frog, and the same, or the opposite sciatic nerve, placed on needle electrodes in the secondary circuit of a Kronecker inductorium.

When the undivided sciatic nerve of the same side is stimulated by a single strong induction shock, two muscular responses (action currents) are obtained, the first (maximal) beginning 0.004 sec. after excitation of the nerve, and the second—a much feebler response—succeeding the first at an interval of about 0.02 sec. When the nerve is severed between the exciting electrodes and the spinal cord, the second response cannot be obtained even by the strongest stimulus. The first effect is due to stimulation of the efferent fibres of the sciatic nerve, and is therefore direct; the second is produced by excitation of the afferent fibres, and is a reflex effect. By deducting the efferent and afferent factors outside the cord from the whole time, it is found that the delay in the cord under normal conditions in the same limb reflex varies in different preparations between 0.012 sec. and 0.022 sec.

When the cord alone has been acted on by a very weak dose of strychnine, the delay is somewhat diminished—0.009 sec. to 0.020 sec.—but when the circulation as well as the cord has been affected by the drug it is increased. Cold applied to the strychnised cord greatly increases the delay; it has the same effect on the normal cord, but to a less degree. Fatigue brought on by repeated stimulation may lengthen the delay in the normal cord.

In the normal cord the crossed reflex (response from one gastrocnemius when stimulus is applied to opposite sciatic nerve) could not be elicited, but in strychnine preparations from which this response can be obtained the delay is about double what it is when the nerve of the same side is stimulated.

It is inferred that in the same-limb reflex there is normally a single synapse interposed in the conducting path, whereas in the crossed reflex there are two such synapses within the cord.

SUTHERLAND SIMPSON.

TEMPERATURE AND EXCITABILITY. KEITH LUCAS and G. R. (122) MINES, *Journ. Physiol.*, Vol. xxxvi., Nos. 4 and 5, p. 334.

THE effect of temperature on the excitability of nerve and muscle in the toad have been studied in relation to electrical currents of long and of short duration. In summing up, the authors conclude that alike for muscle and nerve “the cooler tissue requires less current-strength for its excitation when currents of long duration are used, but when short currents are used this effect is masked, either partially or completely, by the greater increase of current-strength which the cooler tissue demands for a given decrease of current duration.”

SUTHERLAND SIMPSON.

**ON THE CONTRACTION OF MUSCLE, CHIEFLY IN RELATION
(123) TO THE PRESENCE OF "RECEPTIVE" SUBSTANCES.**

Part I. J. N. LANGLEY, *Journ. Physiol.*, Vol. xxxvi., Nos. 4 and 5, p. 347.

IN a previous paper facts had been brought forward by the author which seemed to prove that both nicotine and curari combine with a "receptive" substance in muscle, i.e. with some substance which is not the actual contractile molecule, though capable of acting upon it, and that these poisons have no special action on motor-nerve endings, most of the functions usually attributed to such nerve endings being really functions of the receptive substances of the muscle.

Subsequently, by further investigation, he brought forward reasons for supposing that in frog's muscle there are at least two respective substances present in the muscle fibre capable of causing a slow contraction—one in the region of the nerve ending, the other in the general substance throughout the muscle fibre; and also two receptive substances similarly related in distribution causing a quick contraction.

In the present communication this work has been continued on the muscles of the frog and additional facts adduced in support of the above-mentioned hypotheses.

SUTHERLAND SIMPSON.

**THE FREEZING OF FROG'S NERVE, WITH SPECIAL REFER-
(124) ENCE TO ITS FATIGABILITY.** JOHN TAIT, *Quar. Journ.*

Exper. Physiol., Vol. i., No. 1, p. 79.

THE author froze a short length of the sciatic nerve of a frog's gastrocnemius nerve-muscle preparation, and observed (1) changes during freezing, and (2) changes during thawing, including fatigue changes.

During the process of freezing, which the author compares with drying of the nerve, conductivity gradually disappears, the immediate onset of freezing being, however, accompanied by a temporary improvement in function. As a rule freezing of the nerve induces convulsive twitching of the attached muscle, but not in cases where the ordinary temperature of "cold rigor" of the nerve lies above the freezing-point.

In the return of conductivity after thawing various stages can be traced. Some considerable time elapses before the nerve functions normally again, and a tendency to "cold rigor" at relatively

high temperatures persists for a time after thawing. In the stage of return of conductivity the nerve is readily fatigued by rapid rhythmical stimulation, and the fatigue thus induced may persist for some minutes.

SUTHERLAND SIMPSON.

PATHOLOGY.

PATHOLOGICAL ANATOMY OF PERIPHERAL FACIAL PARALYSIS AND OF FACIAL HEMISPASM. (Contribution à l'étude de l'anatomie pathologique de la paralysie faciale périphérique et de l'hémispasme faciale.) ANDRÉ THOMAS, *Rev. Neurol.*, déc. 30, 1907, p. 1273.

THOMAS has examined the facial nerve and its nucleus in three cases of peripheral facial paralysis, two of which were accompanied by hemispasm.

In the first case the paralysis was preceded by an intercostal herpes zoster, and was followed in eighteen days by a fatal termination from congestion of the lungs. The nerve showed a pure parenchymatous degeneration extending downwards from the first bend in the aqueduct of Fallopius, below which point there was an almost entire absence of axis cylinders. Above the geniculate ganglion the axis cylinders reappeared, were for the most part swollen, and gradually became more normal as the nucleus was approached. The cells of the nucleus were swollen, showed chromatolysis and eccentric nuclei. The nucleus of the opposite side was perfectly normal.

In the second case a total left facial paralysis supervened upon a suppurative otitis media. It was accompanied by loss of hearing, diminution of taste in the anterior part of the tongue, complete reaction of degeneration and slight intermittent spasms of the muscles. Examination of the nerve revealed a chronic perineuritis without interstitial inflammation of the nerve, situated just below the first bend of the aqueduct of Fallopius. The nerve at this level and below it showed a neuroma of regeneration. The geniculate ganglion was somewhat atrophied. The intra-pontine portion of the nerve and its nucleus were normal.

In the third case there was an almost complete right facial paralysis with occasional spasms affecting all the muscles supplied. The peripheral branches showed no degeneration, but many fine fibres twisted and tangled amongst each other, resembling fibres in process of regeneration. Several fine fibres might be seen within a single sheath of Schwann. At the level of a swelling of the nerve at the first bend of the aqueduct of Fallopius, Cajal's silver method

showed numerous fine fibres twisted on each other, suggesting a regenerative process. Below this swelling there was neither perineuritis nor endoneuritis, but the nerve showed traces of a process of irritation as well as indications of restoration. The nucleus of the nerve was in all respects normal.

The authors draw special attention to the fact that in these two cases, where the paralysis was complicated with hemispasm, there was a neuroma of regeneration in the neighbourhood of the geniculate ganglion. They do not express an opinion as to whether the hemispasm is the result of the irritation of compression produced by the neuroma, or is due to some other cause.

It is interesting to note that in three cases of apparently different types of facial paralysis the initial lesion was situated in the aqueduct of Fallopius at the point of the first bend of the facial nerve.

ALEXANDER BRUCE.

HETEROTOPIA OF THE NUCLEUS ARCUATUS. (Ueber einen (126) früheren Fall von Heterotopie des Nucleus arcuatus). M. OECONOMAKIS, *Neurolog. Centralbl.*, 16 Dec. 1907, S. 1158.

THE author describes a medulla oblongata in which the one pyramid was divided up by a very large and branching arcuate nucleus, which was normal in structure. The only interest in the case is, that corresponding to the unusual size of the arcuate nucleus the fibræ arcuatæ externæ ventrales of the same side were unusually large and seemed to be intimately connected with the nucleus.

GORDON HOLMES.

ON THE FORMATION OF NEUROGLIA PENCILS AND (127) ATROPHY OF THE ANTERIOR HORNS IN THE SPINAL CORD OF A GENERAL PARALYTIC. (Ueber Gliastiftbildung und Vorderhornatrophie im Rückenmarke eines Paralytikers.) E. FRIEDEL, *Monatsschr. f. Psych. u. Neur.*, Bd. 22, Ergänzungsheft, p. 39.

THE clinical and anatomo-pathological report of a case of general paralysis with old infantile paralysis. The examination of the cord showed marked wedges of glial hypertrophy, especially in relation to the peripheral neuroglia in the region of Lissauer's zone.

C. MACFIE CAMPBELL.

NOTE ON CELL-FINDINGS IN SOFT BRAINS. E. E. SOUTHARD (128) and M. D. HOSKINS, *Amer. Journ. Insan.*, Vol. xxiv., Oct. 1907.

THE authors call attention to a point in pathological anatomy, viz., the occasional occurrence at autopsy of brains and cords which

are unduly soft to the touch, but which have been subjected neither to œdema nor to post-mortem autolysis. This very diffuse malacia appears to be a phenomenon of terminal exhaustion, and is perhaps related to such central neuritis as may occur (1) in Korsakow's psychosis, or (2) in other conditions, *e.g.* epilepsy. The histology of the brain and cord in œdema and post-mortem autolysis differs from that of the general encephalo-malacia under discussion, as the latter condition shows axonal reacting in the nerve cells and Marchi degeneration in the nerve fibres. As an example of the condition the case of an epileptic dying at the age of forty-two is fully reported.

D. K. HENDERSON.

DIFFUSE CORTICAL CHANGES IN CASES OF CEREBRAL

(129) **TUMOURS.** (Über diffuse Hirnrindenveränderungen bei Hirntumoren.) E. REDLICH, *Arb. a. d. Neurol. Instit. a. d. Wien Univ.*, Bd. 15, Teil 1, S. 320.

THIS paper deals with the psychical changes which are so frequently met with in cases of cerebral tumour, and the question of whether they can be correlated with changes found in the cerebral cortex. After considering the work and opinions of other writers on the subject, the author describes the changes seen in four cases of cerebral tumour accompanied by marked psychical disturbances. The four cases were respectively metastatic carcinoma, glioma, multiple tubercle, and endothelioma of the dura mater; in all, the changes were much alike—slight thickening of the pia mater, with some lymphocyte infiltration, but no plasma cells, loss of granules in the ganglion cells, increase of the satellite cells, especially in the large pyramidal cell stratum of the cortex, neuronophagy, some degeneration of, particularly, the short association fibres, and slight changes in the vessels. These changes are very similar to those met with in senility and in various psychoses, and cannot be regarded as characteristic of brain tumours. The author's opinion is that they are probably due, not to any toxæmia, but to the increase of intracranial pressure and interference with the circulation and nutrition of the cortex.

J. H. HARVEY PIRIE.

EXPERIMENTAL LESIONS AT THE BASE OF THE BRAIN.

(130) (Über experimentelle Läsionen an der Gehirnbasis.) A. SPITZER and J. P. KARPLUS, *Arb. a. d. Neurol. Instit. a. d. Wien. Univ.*, Bd. 16, T. 2, 1907, p. 348.

LESIONS were made in the pons for the most part in cats, dogs and monkeys, and the resulting degenerations traced by the Marchi

method. The method of operating is given in great detail. They reached the pons from in front, entering through the basisphenoid. Sepsis followed in some of the cases. Various fibre systems, afferent and efferent, were involved in the lesions, and it was observed that the physiological effects of the interruption of important anatomical bundles were often very slight. Extensive injury to the pyramidal tracts led to no noteworthy disturbance in cats and dogs, a fact which is in accordance with the findings of several earlier observers. In one of the monkeys the pyramidal fibres were almost all cut across in the pes pedunculi with the involvement of neighbouring bundles, and there was no very evident hemiplegia as a result. This is supplementary to the work of Rothmann and of Schüller, who divided the pyramidal tract at the decussation and on the spinal side of the pons respectively.

In this paper the anatomical results are given for the monkey alone, those for the cat and dog being reserved for a subsequent publication, in which the authors will sum up and correlate the facts of the completed work.

SUTHERLAND SIMPSON.

CLINICAL NEUROLOGY.

DIPHTHERITIC PARALYSIS. (*Les Paralysies diphtériques.*)
(131) CHÉNÉ, *Gaz. des Hôp.*, Jan. 18 and 25, 1908, pp. 75-82 and 111-118.

A LENGTHY review containing no original observations. Too much importance is attached to the treatment of paralysis by antitoxin, the value of which the writer, like many of his compatriots, wrongly assumes is firmly established. (*v. Rev. of Neur. and Psych.*, 1905, p. 129, and 1907, p. 862.) A complete bibliography of recent work is appended.

J. D. ROLLESTON.

DIPHTHERITIC NEURITIS OF THE LEFT CIRCUMFLEX
(132) NERVE. (*Névrite toxique d'origine diphtérique, localisée au nerf circonflexe gauche.*) ESPRIT, *Le Dauphiné Médical*, Dec. 1907, p. 273.

A YOUNG soldier had an attack of diphtheria in September 1907, which was treated with antitoxin, and followed by paralysis of the palate. At the beginning of November, he began to suffer severe pain, especially at night, in the left shoulder. There was no joint trouble nor any history of injury. The unusual prominence of the acromion and head of the humerus with flattening of the shoulder

simulated a forward dislocation of the head of the humerus. The latter, however, occupied its normal position, and the phenomenon was found to be due to atrophy of the deltoid in which the reaction of degeneration was present. Abduction and elevation of the left arm were impossible. The other muscles of the arm and scapulo-thoracic region were unaffected with the exception of the pectoralis major, which was slightly atrophied. The sensory troubles were mainly of a subjective character. Tactile anæsthesia and thermo-anæsthesia were absent nor were there any vasomotor or trophic disturbances. The issue of the case is not recorded.

J. D. ROLLESTON.

PERIPHERAL FACIAL PALSY. ALFRED FUCHS, *Arbeit a. d.* (133) *Neurolog. Instit. a. d. Wien. Univ.*, Bd. 16, Teil 2, S. 245.

THIS paper, as its sub-title states, is a comparison of the more recent literature with the author's personal experiences. Its nature is such that an abstract can scarcely be made, consisting as it does largely of brief abstracts of many cases of peripheral facial palsy of every variety. It is valuable, if only for the literature references.

J. H. HARVEY PIRIE.

THE RECENT EPIDEMIC OF POLIOMYELITIS. V. P. GIBNEY (134) and CHARLTON WALLACE, *Jour. Am. Med. Ass.*, Dec. 21, 1907, p. 2082.

THIS paper is in the form of a preliminary report on cases seen at the Hospital for Ruptured and Crippled during the epidemic in New York last summer. It does not contain much that is new. The occurrence of the epidemic in summer suggests infection by the intestinal tract, and some 63 per cent. of the cases had intestinal disturbances of some sort before the onset of paralysis. Mild cases were seen where the paralysis cleared up practically entirely; others, where a considerable involvement of the meninges had evidently occurred, a point of interest in view of the findings of Harbitz and Scheel in the Norwegian epidemic (see this Review, Feb. 1908, p. 99). Under treatment they lay great stress on early orthopædic measures for the prevention of deformities. The limbs should be kept in normal position, no active treatment applied till the inflammatory changes in the cord have subsided, and even then care must be taken not to overstrain the partially paralysed muscles, or deformities will result.

J. H. HARVEY PIRIE.

**SYRINGOMYELIA WITH BULBAR PHENOMENA AND INTENSE
(135) TROPHIC DISTURBANCES.** RAYMOND and LEJONNE, *Nouv.
Icon. de la Salpêtrière*, July-August 1907, p. 261.

IN this case the motor symptoms consisted of a spastic paraplegia and a paresis of the right arm. The sensory symptoms were those of a hypo-æsthesia to all forms of cutaneous sensation on the right half of the body, as far as the neck; over the left shoulder the temperature sense was lost. Deep sensation was much affected over the right half of the body. Trophic disturbances consisted of a scoliosis, an arthropathy of the right shoulder, and a hypertrophy of the wrist and hand of the same side, with vasomotor affection. The bulbar phenomena were hemiatrophy of the right half of the tongue, paresis of the right half of the palate and of the right vocal cord (syndrome of Hughlings Jackson). The cheiromegaly is of considerable interest because of its rarity. In syringomyelia it is usually one limb, and in particular the peripheral part of a limb, that is affected. If it is bilateral, one side is usually more affected than the other. The cheiromegaly has nothing to do with acromegaly. It is possibly of mixed origin, a combination of hypertrophic osteitis with overgrowth of the soft parts.

S. A. K. WILSON.

A CASE OF SYRINGOMYELIA WITH CHEIROMEGALY. LHER-
(136) MITTE and ARTOM, *Nouv. Icon. de la Salpêtrière*, Sept.-Oct. 1907,
p. 374.

IN a typical case of syringomyelia the interesting feature is the hypertrophy of the right hand, wrist, and lower part of the forearm. Cheiromegaly is usually easy to distinguish from acromegaly. In the former, the length of hand and fingers is not nearly so much affected as the breadth or width; of the latter Marie says that there is always proportion in the disproportion. In the former there is very frequently some affection of the skin, some callosity or fissure, or scar of a blister or whitlow, which is absent in the latter. Osseous change is more regular and more common in the latter than in the former. The pathological anatomy of cheiromegaly is not satisfactorily known. Peripheral neuritis of the nerves supplying the hand, and a syringomyelic cavity more developed in the right than the left half of the cord, were found in the present case.

S. A. K. WILSON.

**A CASE OF PARAPLEGIA DUE TO AN INTRA-MEDULLARY
(137) LESION, AND TREATED WITH SOME SUCCESS BY THE
REMOVAL OF A LOCAL ACCUMULATION OF FLUID.**

WARRINGTON and MONSARRAT, *Lancet*, Jan. 11, 1908, p. 94.

THE salient features of this case were: injury to the lower part of the spine in a man of twenty-two, followed by pain in the lower limbs and trunk for two years; a slow and slight weakness in the legs, becoming worse after a period of rest in bed; and, after an attack of pneumonia, a gradual recovery; then a sudden complete paralysis of the right leg and a gradual paralysis of the left, leading to complete paraplegia. No evidence of syphilis or tuberculosis. Clinically he presented the signs of a transverse lesion of the cord at about the mid-dorsal region, the absolute loss of power, great loss of sensation, and the feebleness of the reflexes showing that conduction was greatly interfered with. The probable diagnosis seemed to be a gliomatous growth consequent on the trauma, with a hæmorrhage into it, accounting for the sudden accentuation of symptoms nearly four years after the injury. At operation the laminae of the 6th to 9th vertebrae were removed, a tough pellicle was found outside the dura, evidently of inflammatory origin. The dura was adherent to the cord, which for some distance was of a jelly-like, greyish-black colour, and protruding. Incision in the posterior mid-line allowed a few c.c.'s of clear fluid to escape. The wound was then closed. Four months later the patient was able to walk a little with crutches, the reflexes were still exaggerated, sensation had greatly improved in the right leg and thigh, but had diminished in degree over an area corresponding to the extent of the surgical procedure.

J. H. HARVEY PIRIE.

**PERIPHERAL SPINAL DEGENERATION REVEALED ONLY BY
(138) LONGITUDINAL SECTION OF THE CORD AND AN AXIS
CYLINDER STAIN. S. D. LUDLUM, *N.Y. Med. Jour.*, Dec.
21, 1907, p. 1167.**

A WOMAN had acute joint inflammation and, three days before death, delirium, but no definite motor nor sensory symptoms. Post-mortem there was found a moderate degree of meningitis, especially of the cord, but the interest of the case lies in the fact that although Marchi and Weigert stains gave negative results, Bielschowski's method applied to longitudinal sections demonstrated a band of degeneration and breaking up of the axones about the entire periphery of the cord. The author thinks this has been due to the contiguous meningitis.

J. H. HARVEY PIRIE.

MENINGITIS FOLLOWING GONORRHOEA. (*Gonocoque et meningococque.*) MILHIT et TANON, *Presse Médicale*, Jan. 15, 1908, p. 34.

A MAN, aged 21, was admitted to hospital on October 18, 1906. Three weeks previously he had contracted gonorrhœa, which ran a normal course till October 15, when the discharge suddenly stopped. The next day, his legs were weak, and on the seventeenth, he was unable to stand. At the same time he suffered from headache and dyspnoea. On admission, though unable to stand, he could still move his limbs. The reflexes of the lower limbs were diminished. There were no sensory nor sphincter troubles, and no mental disturbance. There was no purulent urethral discharge, but serous fluid expressed from the meatus showed a few extra-cellular gonococci. On lumbar puncture the cerebro-spinal fluid was found to be normal. On October 23, the paralysis invaded the upper limbs and the diaphragm. The headache became worse, and there was stiffness of the neck and of the whole vertebral column. The reflexes of the upper and lower limbs were weakened but not abolished. Temperature 102.2°. Lumbar puncture was again performed. The cerebro-spinal fluid was now turbid, and on centrifugalisation showed polynuclears containing a few encapsuled diplococci, which closely resembled Weichselbaum's organisms. The next day coma supervened, and complete palsy of the limbs. The sphincters were paralysed, 20 c.c. of very turbid cerebro-spinal fluid were withdrawn under tension. Numerous polynuclears, pus cells, and meningococci were found. The following day there was improvement which was subsequently maintained. On November 1, commencing muscular atrophy was noted, starting symmetrically in the lower limbs and invading the abdominal muscles, thorax, and upper limbs. There was no reaction of degeneration. Massage, electricity, and tonics were employed, and the atrophy gradually disappeared. Complete recovery took place.

J. D. ROLLESTON.

ACUTE SYPHILITIC MENINGITIS. (*Méningite aiguë syphilitique.*) (140) LAUBRY et GIROUX, *Tribune Médicale*, Jan. 4, 1908, p. 837.

A WOMAN, aged 24, a dancer by occupation, was admitted to hospital on May 9, 1907, with the following history. Beyond a miscarriage three years previously there was nothing to note in her antecedents. Two months ago she had suffered from attacks of violent headache and lumbar pain preceded or followed by tinnitus, nausea and vomiting. The ordinary treatment for

neuralgia was adopted, and the symptoms so far remitted as to allow her to make a long voyage and to continue her occupation for about a month. On May 2 she complained of vertigo, diplopia, and dimness of vision. Walking became increasingly difficult. She also had intense headache and backache, vomiting, and obstinate constipation. Contracture of the neck and trunk muscles, the presence of Kernig's and Babinski's signs, and right ptosis, showed that the cerebro-spinal system was involved, but the diagnosis of epidemic cerebro-spinal meningitis was set aside by the absence of grave general phenomena, while tuberculous meningitis was negatived by the absence of visceral tuberculosis. Antisyphilitic treatment consisting of mercurial injections and iodide of potassium was adopted. On admission to hospital, the vomiting had ceased for three days, but there was still frontal headache and obstinate constipation. The neck was slightly extended. The knee jerks were exaggerated, and there was slight clonus. Babinski's sign was present on both sides, especially on the left. Kernig's sign was easily obtained. Sensibility was exaggerated. Light and accommodation reflexes were present but sluggish, and there was diplopia with convergent strabismus. The abdomen was retracted. Temperature 100°. Pulse 100.

In the left frontal region was a small tender exostosis, which had hitherto escaped notice. After one week's antisyphilitic treatment, the pyrexia subsided, and the pain in the back and limbs, the strabismus and ptosis disappeared. When discharged on June 28, the patient still had exaggerated reflexes, clonus and bilateral Babinski, but no Kernig. The exostosis had almost entirely disappeared. Lumbar puncture was performed on three occasions, first on admission, when the fluid was clear, slightly yellow, without hypertension, and showed a pure lymphocytosis (10-15 cells in each field). The fluid withdrawn at the second puncture four days later was similar. Neither the tubercle bacillus nor the spirochæta pallida was found. At the third puncture, performed forty days after admission, the lymphocytosis was less (8-10 cells in each field).

J. D. ROLLESTON.

**THE VALUE OF LUMBAR PUNCTURE: WITH PARTICULAR
(141) REFERENCE TO THE DIAGNOSIS OF TUBERCULOUS
MENINGITIS.** EUGENE P. BERNSTEIN. *Mount Sinai Hospital
Reports*, Vol. V., 1907, p. 491.

THE conclusions arrived at are based on the examination of 617 cerebrospinal fluids.

In cases of tumour of the brain several sudden deaths have been reported after the withdrawal of comparatively small amounts of fluid. It is advisable to stop the flow of fluid when

the pressure reaches 20-25 mm. of mercury. This, according to Pfaundler, is the normal subarachnoid pressure in the sitting position, about one-half of which disappears when the patient lies down. The consensus of opinion is that in normal individuals the cerebrospinal fluid usually measures in amount less than 10 c.c.

With regard to the presence of sugar, Comba states:—

1. In the healthy person a glucose-like reducing substance is constantly present.

2. In tuberculous meningitis the glucose is found in small amounts at the onset, but is absent towards the end.

3. In purulent meningitis it is always absent.

The author found in 53 tuberculous fluids that Fehling was reduced in 12, and these reactions all occurred but a few days at most before death. Therefore the presence of this reducing substance does not exclude advanced tuberculous meningitis. At least 10 c.c. of fluid should be used when the glucose-like body is looked for, or its presence may escape detection.

Bernstein has never found the Fehling reducing body in purulent meningitis.

Tuberculous meningitis is almost always accompanied by a mononuclear leucocytosis. In 102 fluids from tuberculous meningitis only 3 fluids proved exceptions to this general rule. Cryoscopy has been found of no value. Negative bacteriological findings are of no importance. The author cites the various organisms which have been met with in the cerebrospinal fluid. In his series the diplococcus intracellularis was the most frequent. Following this the tubercle bacillus. In the 102 cases examined tubercle bacilli were found in no less than 100 (=98 per cent.), although in more than one case it was necessary to search for several hours before their presence was detected. The technic in looking for tubercle bacilli is described in detail. Langar diagnosed 100 per cent. of his cases by cultivation, but unfortunately the patient is usually dead long before the diagnosis is obtainable. Subcutaneous or intraperitoneal inoculation of the cerebrospinal fluid into guinea-pigs is, according to the author, absolutely reliable, his results being uniformly good, even when only one or two c.c. were obtained for the purpose. This method is open to the same objection, however, as cultivation, since four to six weeks must be allowed to elapse before the animal is killed. Four cases of recovery from tuberculous meningitis have been reported in which the tubercle bacilli were demonstrated in the cerebrospinal fluid. All of the author's 102 cases ended fatally.

EDWIN BRAMWELL.

THE TECHNIC OF LUMBAR PUNCTURE IN CHILDREN.

(142) HENRY HEIMAN. *Mount Sinai Hospital Reports*, Vol. V., 1907.

THE author describes in detail the technic which he and Koplik have found from a considerable experience to be most serviceable. In forty lumbar punctures, the patients varying in age from three months to twelve years, the highest pressure directly after insertion of the needle was 54 centimetres, the lowest 4 centimetres, the average 26.2 centimetres. In these same cases the average of the various quantities, which were of necessity withdrawn to render the pressure normal, was 32 c.c., the maximum 60 c.c., and the minimum 5 c.c. In cases of hydrocephalus the author has on three separate occasions drawn off more than 150 c.c. without any ill effects. The causes of "dry taps" are faulty technic, propulsion of the dura before the point of the needle, occlusion of the needle with tissue, fibrin, or pus, abnormally small amount of secretion, or closure of the foramen of Magendie or aqueduct of Sylvius, owing to the pressure of a tumour or to inflammatory conditions.

EDWIN BRAMWELL.

HEMIPLEGIA AS A COMPLICATION OF TYPHOID FEVER.

(143) F. SMITHIES, *Journ. Amer. Med. Assoc.*, Aug. 3, 1907, ii., p. 389.

HEMIPLEGIA in typhoid fever is rare. Smithies has collected only forty-six cases from literature, including the present one. (To these may be added the two cases of Barié and Lian, and Laignel-Lavastine, *v. Rev. of Neurol.* 1908, p. 37. J. D. R.). It is commonest in early adult life, doubtless owing to the greater prevalence of typhoid fever at that age. The time of onset varied widely. In only one did it take place in the first week. The great majority was found in convalescence. Aphasia was noted in twenty-six cases. Right hemiplegia occurred in twenty-one, left in ten. Twenty-three were males, nine were females. Death occurred in about 15 per cent. Smithies' case was that of a farmer who twelve years previously had suffered from syphilis, which he had left untreated. Left hemiplegia, preceded for a few days by cramp in the fingers of the left hand, and tremors of the jaw and upper part of the trunk, developed on the fifteenth day. Considerable improvement took place, but does not appear to have been complete. Severe headache occurred in convalescence, which disappeared after the exhibition of iodide of potassium. The gradual onset and progressive recovery

point to thrombosis of one of the cerebral arteries, probably the right middle cerebral. Syphilis is incriminated as a predisposing cause of the thrombosis.

J. D. ROLLESTON.

BRAIN TUMOUR WITH JACKSONIAN SPASM AND UNI-
(144) LATERAL PARALYSIS OF THE VOCAL CORD, AND
LATE HEMIPARESIS AND ASTEREOGNOSIS. J. T.
 ATLEE and C. K. MILLS. *Journ. Amer. Med. Assoc.*, Dec. 28,
 1907, p. 2129.

THIS case is of interest both from the symptomatology and the character of the tumour. The patient was a physician of 43, who was successfully operated on for a tumour, originating at the lower end of the right precentral convolution, involving a large part of it, and to some extent also the parietal lobe. The tumour was easily shelled out, and was proved to be of the nature of a hyperplasia of the choroid plexus. His symptoms had been Jacksonian spasms of the left side with an initiating sensory aura, the attacks consisting of a sensory discharge confined to the face, tongue, throat, and neck. Later there was weakness and astereognosis of the whole left side and failure of word memory. These phenomena are readily explicable from the site of the tumour. But he had also unilateral (left-sided) paralysis of the vocal cord. This had all the characters of a peripheral recurrent laryngeal affection, and, indeed, it cannot be positively stated that it was not due to such disease (there was some suggestion of aneurism). The voice recovered considerably after the operation, although the cord still remains paralysed. It is generally believed that the cortical laryngeal centres have bilateral control, but this case, and a clinical one of Delavan's, suggest the possibility of a destructive lesion of the cortical laryngeal centre producing a persistent unilateral paralysis of the opposite vocal cord.

J. H. HARVEY PIRIE.

A CASE OF PSEUDO-BULBAR PARALYSIS WITH COMPLETE
(145) LOSS OF VOLUNTARY RESPIRATION. CHARLES E
 BEEVOR, *Arch. a. d. Neurol. Instit. a. d. Wien. Univ.*, Bd. xv.,
 Teil 1, 1907.

THE clinical report of a case of paralysis of all the voluntary movements of the lower part of the face and mouth, and also of the voluntary movements of inspiration and expiration. The condition occurred after three attacks of hemiplegia—two affecting the left side and one the right side—in a man who had contracted syphilis three years previously.

Though all voluntary movements of respiration were lost, the emotional or reflex actions of laughing, crying, coughing, sneezing, and yawning were preserved, and in addition the action of the muscles of the trunk and of the limbs was quite good for movements which were not respiratory. The action of the latissimus dorsi muscle is specially considered, the writer pointing out that it acts as a strong expiratory muscle in sneezing and coughing.

The lesions are supposed to have involved the genu and anterior one-third of the posterior segment of both internal capsules, extending down to the anterior one-third of the crusta, and also the regio subthalamica and the part between the crusta and the middle line, as well as the anterior part of the internal nucleus of the optic thalamus.

D. K. HENDERSON.

A CASE OF PSEUDO-BULBAR PARALYSIS. (*Sur un cas de (146) paralysie pseudo-bulbaire.*) F. RAYMOND et L. ALQUIER, *Nouv. Icon. de la Salpêtrière*, Sept.-Oct. 1907, p. 371.

THE symptoms in this patient, a woman of 77, were, immobility, an almost complete mental confusion, with attacks of laughter or weeping from time to time. In addition, a spastic paresis of all four limbs, exaggerated reflexes with ankle clonus and Babinski sign, and complete incontinence of the sphincters. No actual bulbar troubles, but the speech was slow and scanning; there was nystagmus, and the upper limbs showed an intention tremor. The autopsy revealed the following. In the cerebrum: numerous lacunar areas of disintegration, some as large as a small pea, in the white matter and in the basal nuclei—they had produced a diffuse demyelination in the principal association tracts, particularly the superior longitudinal and the occipito-frontal bundles. There were no lacunes in the internal capsule. The peduncles were also spared. In the pons and upper part of the medulla were numerous lacunar areas, mostly very small, but four exceedingly large ones, one of which involved part of the pyramidal tract. The upper part of the cervical cord also showed some lacunar disintegration, similar to those recorded by Lhermitte in senile paraplegias. One may conclude that the pseudo-bulbar syndrome merely indicates the existence of a lesion above the bulbar nuclear; usually cerebral, it may be pontine. This would be suggested by symptoms such as were present in this case, nystagmus, scanning speech, and intention tremor, the intellectual troubles pointing to the cerebral lesion.

J. H. HARVEY PIRIE.

POST-APOPLECTIC TREMOR. (Symmetrical Areas of Softening in (147) both Lenticular Nuclei and External Capsules.) JOHN H. RHEIN and CHARLES S. POTTS, *Journ. Nerv. and Ment. Dis.*, Dec. 1907.

THE case reported is that of a man aged 58, who, when examined in 1906, had an almost constant movement of the right arm, which only ceased temporarily when his attention was distracted. The movement consisted of alternate flexion and extension at the elbow and wrist, the hand being supinated. There was no loss of power in the arms, but the legs were rather feeble, and the right one somewhat stiff. There was ataxia of both arms and legs, especially noticeable in the right arm. The deep reflexes of the arms were diminished; the knee-jerks, especially the left, were brisk. The plantar reflexes were flexor. Gait was slightly ataxic, and Rombergism was present. The reaction of the pupils to light was sluggish.

The patient's mental condition precluded any concise account of the onset of illness being obtained, but the condition was of long duration (several years), and progressive.

The pathological examination revealed softening in the putamen on each side, more extensive on the left than on the right, also degeneration of the fasciculus of Türck on the right side. There was no degeneration of the motor fibres in the internal capsule, the pons, medulla, or cord. On the right side "the red nucleus appeared to be intact"; on the left side "the red nucleus appeared to be normal." The dentate nucleus on both sides "showed an unusual vascularity."

T. GRAINGER STEWART.

REPORT OF TWO FATAL CASES OF BRAIN ABSCESS. (148) THOMSON, *Arch. of Otol.*, Vol. xxxvi., p. 576.

THIS is a full clinical account of two cases of brain abscess—the first that of a large cerebellar abscess involving the entire left lobe, a portion of the central lobe, and encroaching on the right lobe, and yet giving rise to no characteristic signs of trouble in that region; the second, a case of temporo-sphenoidal abscess with typical symptoms which repeated puncture through the dural exposure in an existing mastoid wound failed to detect. In both cases the indications from the leucocyte count were negative. The first case is chiefly of interest from the fact that such a large destructive lesion could exist in the cerebellum without giving rise to more definite signs of its presence; the second raises the question as to the

relative value of puncture and exploration through the already existing mastoid wound in cases of suspected temporo-sphenoidal abscess, compared with that through a trephine opening over the superior or middle convolution. HENRY J. DUNBAR.

A FATAL CASE OF SINUS THROMBOSIS, AFTER CHRONIC (149) PURULENT OTITIS COMPLICATED WITH CHOLESTEATOMA, ILLUSTRATING AN UNUSUAL VARIETY OF INFECTION. KNAPP, *Arch. of Otol.*, Vol. xxxvi., p. 573.

A BOY of eight years, the subject of kyphosis, who had suffered for three years from discharge from the right ear, was admitted with a three days' history of headache, vomiting, cessation of discharge, and rigors. The temperature was 105° and the pulse 140. There was tenderness over the mastoid, and pain below the mastoid tip, extending down into the neck. At the operation thin foetid pus was found in the dilated antrum and cholesteatomatous masses in the tympanum. The sigmoid sinus contained disintegrated clot, thin serous pus, and gas smelling like an appendix abscess. Two days later there was tenderness in the neck and enlargement of glands. The internal jugular was ligatured; it was inflamed and adherent, but contained fluid blood. Symptoms of septic pneumonia developed, and the patient died in a few days. Clinically and bacteriologically the infective agent belonged to the proteus-aërogenes group, which, in sinus thrombosis secondary to mastoiditis, has chiefly been observed in chronic purulent otitis with cholesteatoma. The jugular vein should have been ligated at the first operation. HENRY J. DUNBAR.

BRAIN TUBERCLE IN CHILDHOOD. (Der Hirntuberkel im Kin- (150) desalter.) J. ZAPPERT, *Arbeit. a. d. Neurolog. Inst. a. d. Wien. Univ.*, Bd. 16, Teil 2, S. 79.

THIS is a long article, containing notes of a large number of cases, with comments thereon. The following is simply a translation of the author's own conclusions or summary:—

1. Brain tubercle in children runs, in the great majority of cases, a latent course; the clinical diagnosis is likewise usually not made.
2. Among those running a latent course may be cases with tumours from the size of a hazel-nut to that of a plum, but in the majority the tumours are small.
3. Multiple tubercular tumours are not so often latent as single are.

4. Tubercles in the cerebellum, cerebrum, and basal ganglia are as often latent as manifest; those in the crura cerebri, pons, and corpora quadragemina usually cause definite symptoms.

5. Latent brain tubercle may give rise to a terminal tubercular meningitis with the onset of localising symptoms of an atypical meningitis.

6. Latent cases may become rapidly fatal with meningeal symptoms without any meningitis being present.

7. The course of a case may run entirely under the clinical picture of a progressive hydrocephalus.

8. General tumour symptoms are not infrequently present without characteristic localising symptoms.

9. In multiple tubercle one of the tumours may be localised with more or less certainty, while the others, even large tumours, have been unsuspected.

10. Localisable tumours are mostly large, and are found especially in the pons, the cerebellum, and the corpora quadragemina.

11. Through the frequent onset of an end meningitis, a limit is set to the growth of the tubercular tumours. Where there is no meningitis, or if only late in the disease, the tumours more often become localisable.

12. Among the initial symptoms the more important are hemiplegia coming on with convulsions and tremor on one or both sides, also ataxia and chorea.

13. The differential diagnosis must be from infantile cerebral paralysis, epilepsy, meningitis, encephalitis, and hydrocephalus.

14. Little good can be looked for from operative interference.

J. H. HARVEY PIRIE.

GLIOTIC CYST OF THE RIGHT SUPERIOR PARIETAL LOBULE.

(151) A. N. COLLINS and E. E. SOUTHARD, *Amer. Journ. Insan.*, Vol. xxiv., Oct. 1907.

THE clinical and anatomo-pathological report of a case showing general and focal symptoms which terminated fatally after lasting for six years. At autopsy a cyst was found 2.5 cm. in diameter which reached to the lateral ventricle, and was only separated from it by the ependymal lining. Fibrous bands projected in all directions throughout the cyst, and seemed to form a more or less intimate part of it. The explanation offered is that of glioma with cystic degeneration, the condition being analogous to syringomyelia.

D. K. HENDERSON.

A STUDY OF CERTAIN REFLEXES IN SCARLET FEVER.

(152) C. ROLLESTON, *Quar. Journ. of Med.*, Jan. 1908, p. 117.

THE writer examined the plantar, abdominal, cremasteric, and knee jerk reflexes in 175 cases of scarlet fever. 75 were male and 100 female.

The cases were divided according to their severity into three classes. Severe (20 cases), moderate (132 cases), and mild (23 cases).

The average age of the patients was $6\frac{1}{2}$ years. The reflexes in children under two were not examined, owing to the infantile character of the plantar and the uncertainty of the abdominal responses. It was found that the severer the case, and the younger the patient, the more likely were the plantar flexor responses to be replaced by Babinski's sign, and the abdominal reflexes to be absent. The knee jerks were found absent in only three severe cases. The cremasteric reflex was only absent in one case.

Of the 20 severe cases 14 exhibited an extensor plantar response, the average duration of this phenomenon being 24 days. The abdominal responses were absent in 13 of the 20 cases, the period of absence extending over 14 days.

Among the 23 mild cases only 4 showed an extensor response, the average duration being 11 days. In 8 of these 23 patients, the epigastric and abdominal reflexes were either absent or diminished. Sixty-six of the moderate cases exhibited Babinski's sign, the average duration being 10 days. In 50 of these 132 cases, the abdominal reflexes were absent, the period of absence being 18 days. In a large number of cases in all three groups, whatever the type of plantar response first obtained, there was an interval of varying duration, in which the plantar reflexes were affected, being either entirely absent, or represented by reaction in the hip muscles alone.

The epigastric and abdominal were less frequently affected than the plantar reflexes. The epigastric was usually affected contemporaneously with the supra- and infra-umbilical responses, but occasionally the epigastric lagged behind the others in its return to activity. Frequently, but by no means always, the absence of the abdominal reflexes was associated with Babinski's sign.

The onset of complications, *e.g.* uræmic fits and endocarditis, was found to alter the character of previously normal reflexes.

AUTHOR'S ABSTRACT.

GRAPHIC STUDY OF FOOT-CLONUS AND ITS SIGNIFICANCE

(153) IN PRACTICE. (Das graphische Studium des Fussclonus und seine Bedeutung in der Klinik.) ETTORE LEVI. *Arch. a. d. Neurol. Instit. a. d. Wien. Univ.*, Teil 2, 1907

THE author of this paper studied the curves obtained by the graphic registration of the movements in foot-clonus, in such

conditions as ordinary hemiplegia, spastic post-puerperal paraparesis, pseudo-bulbar paralysis, multiple sclerosis with hysterical complications, hystero-neurasthenia, as well as in normal individuals after fatigue.

He found that the periodicity of the movements in the purely organic lesions lay between 4 and 7 per second, being generally about 6 per second. The rate was never found to be so high as that recorded by Horsley (8-10) or MacWilliam (13·5-14), and it did not vary during the course of an experiment, though it differed slightly from day to day according to the state of the patient as regarded fatigue. The extent of the organic lesion seemed to have no influence on this rate, which was independent of the will of the individual. The single elements of the tracings showed regularity of form as well as of rhythm.

In cases of pseudo-clonus the periodicity was found to vary from second to second, reaching a maximum of 5-7 per second. The single movements showed irregularity in extent, some appearing "half abortive."

The author thinks that the differences between these two types of curves are sufficiently clear to be of clinical importance. He also found that after hard exercise of the lower extremities in healthy individuals (*e.g.*, after hard bicycle exercise on hilly roads), a foot-clonus could be induced which gave a curve like those got in pseudo-clonus.

J. GRAHAM BROWN.

OCULO-MOTOR PARALYSIS WITHOUT INVOLVEMENT OF (154) THE INTERNAL MUSCLES IN PERIPHERAL LESIONS.

(Oculomotoriuslähmung ohne Beteiligung der Binnenmuskeln bei peripheren Läsionen.) FUCHS, *Arch. a. d. Neurol. Instit. a. d. Wien. Univ.*, Bd. 15, T. 1, 1907, S. 1.

THE author states that the opinion that in a pure external ophthalmoplegia the lesion is to be found only in the oculo-motor nuclei, or in the intra-cerebral course of the nerves, is not invariably true; that, when a typical external ophthalmoplegia is bilateral, its origin is usually nuclear; but, on the other hand, when the ophthalmoplegia is unilateral it is probably not nuclear. A purely unilateral ophthalmoplegia or a paralysis of the third nerve without involvement of the interior muscles has been recently shown to be a possible result of a peripheral basal lesion. The author gives references to eighteen such cases, in six of which section showed compression atrophy, primary atrophy of the nerve itself, and inflammations. He discusses in detail five clinical observations of his own.

The power of causing a pure external paralysis is peculiar to no

special kind of lesion, but may be observed in trauma, such as from fracture of the base of the skull, or from inflammation, as in neuritis, meningitis or orbital cellulitis, or from simple atrophy or atrophy due to compression. The writer attributes the escape of the internal muscles to a greater resisting power of their nerves, and refers (1) to the opposite condition of diminished resisting power in the macular fibres of the optic nerve which, in spite of their central position, are most easily affected in cases of a retro-bulbar neuritis, intoxications, etc. ; and (2) to the frequent escape of the centripetal pupil fibres, where not infrequently in neuritic atrophy of the first nerve, the light reaction of the pupils is retained after the sensibility to light is lost. These two conditions prove that different parts of a nerve trunk may have different degrees of vulnerability, and afford a reasonable explanation for the escape of the internal muscles of the eye, in some cases when the peripheral part of the oculo-motor nerve is paralysed.

ALEXANDER BRUCE.

**THE BACTERIOLOGICAL ASPECTS OF THE PROBLEM OF
(155) NEUROPATHIC KERATITIS.** H. MORRISTON DAVIES and
GEORGE HALL, *Brit. Med. Journ.*, Jan. 11, 1908.

IN this paper the authors call attention to the absence of bacteriological investigations in the work previously done on the causation of neuropathic keratitis. While they do not claim that the solution of the problem has been made out, they reached the conclusion that this mode of attack is decidedly more hopeful than those formerly used.

The first part of the paper consists of a detailed review and criticism of the various existing hypotheses concerning the etiology of the disease.

The second part deals with the bacteriological investigations. The authors made repeated cultures from the eyes of twenty-one patients who had had the Gasserian ganglion removed. Nine of these were seen during an attack of neuropathic keratitis, whilst ten patients (seen twelve days to eighteen months after the operation) had never had any eye trouble. The organisms obtained from the cultures were staphylococcus aureus and albus, streptococcus, pneumococcus, xerosis and proteus, together with one other bacillus, which belongs to the group of pseudo-diphtheria bacilli, this organism the authors refer to as the *Bacillus X*. The staphylococcus aureus was obtained from every eye examined, whilst the *Bacillus X* was found in all the eyes, and in those only, which were affected by neuropathic keratitis.

The experimental work on eight monkeys was performed by Sir Victor Horsley. The following results were obtained :—In three

monkeys the cornea was not perfectly anæsthetic after the operation, and no keratitis developed either spontaneously nor after inoculation with *Bacillus X*. In three others the *Bacillus X* was present, and the keratitis appeared within twenty-four hours of opening the lids. In one no keratitis could be induced after the operation, but within twenty-four hours of inoculation the *Bacillus X* was never recoverable, only staphylococci being found, whilst in one rhesus well-marked neuropathic ulceration was obtained after inoculating the conjunctiva with the discharge from the eye of another monkey suffering from keratitis. This discharge contained staphylococcus aureus and *Bacillus X*. The coccus was found to be non-pathogenic to guinea-pigs.

	Number of cases examined.	Presence of staphylo- coccus.	Presence of <i>Bacillus X</i> .
Cases with neuropathic keratitis—			
Acute stage	9	9	9
Quiescent stage	6	6	1 ¹
Cases without neuropathic keratitis	9	9	0

¹ The case developed another attack of keratitis later.

The investigations suggest to the authors that three factors are necessary: (1) Removal of the Gasserian ganglion; (2) presence of the bacillus; (3) a factor of undefined nature dependent on the eyelids and removed by closing them. AUTHOR'S ABSTRACT.

AMAUROTIC FAMILY IDIOCY. (*Tay-Sachs Disease.*) (*L'Idiotie (156) amaurotique familiale.*) M. E. APERT, *Semaine Méd.*, jan. 15, 1908.

BEING led to study amaurotic family idiocy by the observation of a case, the first published in France, the author in this article arrives at the following conclusions:—

1. Amaurotic family idiocy is a well-defined morbid entity, almost identical in the great majority of published cases, and characterised by the following peculiarities—onset during the first six months of life, with a progressive apathy; gradual enfeeblement of the power of sight; pathognomonic alteration of the fundus of the eye consisting in a regularly circular white spot situated in the macular region, with a cherry-red point in the centre; emaciation; paralysis; idiocy; marasmus; death generally before the age of two. On autopsy, no macroscopical alteration of the nervous

centres. On histological examination, a special translucent, globular degeneration of the cells of the nervous centres and of the ganglion cells of the retina. The disease has a very distinct family character; in numerous families about half of the children are affected one after the other. The predilection of the disease for the Jewish race is remarkable; taking into account only cases which present in a typical way the characteristic picture described above, out of eighty-two observations two only are those of non-Jewish children.

2. Besides the typical form characterised by such special lesions of the macula, there exist some near varieties, in which the morbid picture is almost the same, but in which there is no white macular spot, but simply variable alterations of the fundus of the eye, usually with lines of pigment. These abnormal forms are less peculiar to the Jewish race than the typical form, since out of nine cases, two only were Jews. In the absence of autopsy, it is difficult to say if these atypical varieties should be identified with the Tay-Sachs disease.

3. Vogt has also recently allied to the Tay-Sachs disease a certain number of observations of family diplegia with amaurosis, for which he proposes the name of "juvenile form of amaurotic family idiocy." But these observations, which for that matter are not all comparable to each other, differ from Tay-Sachs' disease in numerous ways: onset during the second period of childhood, spasmodic paraplegia, absence of the pathognomonic alterations in the fundus of the eye, and, in the rare cases in which there was autopsy, granular and pigmentary alteration of the brain cells quite different from the globular infiltration by a translucent substance which is constant in Tay-Sachs' disease. We must therefore continue to class these cases apart under the name of family diplegia with amaurosis (Higier). By doing so we shall retain for amaurotic family idiocy the character of a morbid entity, so well defined by its constant pathognomic characters and its precise limits, as they have been traced by the first observers of the disease.

AUTHOR'S ABSTRACT.

**OTALGIA CONSIDERED AS AN AFFECTION OF THE SENSORY
(157) SYSTEM OF THE SEVENTH CRANIAL NERVE. HUNT,
Arch. of Otol., Vol. xxxvi., p. 543.**

THE distribution of the pain in herpes zoster auris, which is dependent on a specific inflammation of the geniculate ganglion, first drew the author's attention to the connection between otalgia and the sensory system of the facial nerve. The pain in these cases is variously referred to the surface of the auricle, the auditory canal or the depths of the ear—the same, approximately, as in idiopathic

and reflex otalgia. Developmentally there is a most intimate relationship between the facial nerve and its ganglion, the ganglia of the acoustic nerve, and all those structures which go to form the auditory mechanism. Anatomically the facial is a mixed nerve, the geniculate ganglion, which is the homologue of the Gasserian ganglion, having as its sensory root the nerve of Wrisberg, and as its peripheral branches the two superficial petrosal nerves, the external petrosal nerve, and other filaments, by means of which the 7th nerve establishes sensory relationships with the internal, the middle and the external ear. In reflex otalgia the lesion giving rise to the condition is observed to be in the distribution of the second or third divisions of the trifacial nerve, which are both anatomically connected with the geniculate ganglion by means of the great superficial petrosal nerve which passes to Meckel's ganglion and the small superficial petrosal which passes to the otic ganglion. Because of the complexity and overlapping of the auditory innervation, certain mixed forms of otalgia occur. These belong rather to the auriculo-temporal neuralgia of the trigeminus, or are occipito-cervical otalgias; not, however, otalgia in the pure sense of the term. The writer concludes by saying that, owing to the numerous and intimate central and peripheral connections of the 5th, 7th, 8th, 9th, and 10th nerves, many careful clinical analyses will be required before deciding the exact role played by any one of them in otalgia; but that it seems clear that while the facial nerve may be by no means the sole factor, it is the preponderating one in the production of otalgia.

HENRY J. DUNBAR.

THE PSYCHICAL DISTURBANCES IN SYDENHAM'S CHOREA.

(158) (Über die psychischen Störungen bei der Chorea minor.)

KLEIST, *Allg. Zeitschr. f. Psych.*, lxiv., 1907, p. 769.

THE author has observed 154 cases of chorea minor (including chorea gravidarum) occurring in the University Clinic at Halle, and has paid special attention to the relation of the chorea to psychical disturbance.

As far as the physical symptomatology of chorea minor is concerned, emphasis is laid on the clonic nature of the movements, their sudden rise and fall: sometimes they are of abrupt onset, but persevere in a tonic way before slowly disappearing. The curve of this type of involuntary movement shows a sudden rise, with a plateau and a gradual subsidence. The distribution of the movements is highly variable; in severe cases the extensors of the back are commonly involved, and a condition not at all unlike the "*arc de cercle*" of hysteria is frequently seen. Inco-ordination, apart from the involuntary element in the disturbance of motility,

is interesting. It is to be observed chiefly in the fingers ; in some severe cases there may be general inco-ordination in standing, walking, sitting, etc. Speaking, eating, swallowing may be impaired. There is no necessary parallelism between the inco-ordination and the twitching ; the former may persist after the disappearance of the latter. The inco-ordinate cases are a link between ordinary chorea and so-called "acute ataxia," in which isolated choreic movements may occur. There is much evidence to suggest that functional disturbance of the cerebellum (inco-ordination and involvement of trunk musculature) plays its part in chorea.

Förster distinguishes between affection of the prime-movers (agonists) and affection of the synergic and antergic muscles in chorea. According to him the former are less impaired than the latter. Kleist finds that the inco-ordinate cases reveal a disturbance chiefly of the prime-movers ; their innervation is fleeting, unsteady, sometimes slow, sometimes feeble. Thus there is a link here with cases of "paralytic" chorea. Many of the cases observed exhibited general weakness of one-half of the body. In two cases of almost complete right-sided paralysis the left side showed great defect in agonist-innervation. The akinesis of severe cases of chorea is not a paresis in the ordinary sense, inasmuch as certain movements may be possible one minute and impossible the next ; nor is it directly proportional to the weakness of the musculature, nor attributable to asynergy. In cases of akinesis there is great diminution of spontaneity, which may persist after the twitching phase is over : automatic movements, movements of expression and mimicry, are often reduced ; there is a curious poverty of expression sometimes. Choreic patients tire readily with muscular effort.

Hypotonia of the muscles is a constant feature. Again, no parallelism between the hypotonia and the involuntary movements is discoverable. The former frequently persists after movements cease. On the other hand, hypotonia and inco-ordination are closely associated. The tendon reflexes are absent, or normal, or increased. Tonic perseveration of the reflex is not infrequent : it has been observed at elbow and ankle as well as at knee. There is sometimes disturbance of sensation, either in the form of pains and paræsthesiæ, or (rarely) in the form of objective change. The muscular sense is always intact. Sometimes nerve trunks are sensitive to pressure. Cutaneous reflexes are frequently elicited with minimal stimuli. In two cases Babinski's extensor response was present.

In 13 per cent. of the 154 cases no obvious psychical impairment was discoverable. In 92 cases there was some mild psychical disturbance, which took the shape of an easily excited anxiety or

timidity, or of childishness, or of diminution of spontaneity. Some of the patients were almost stuporose. In most instances the actual choreic phenomena and the altered psychical states ran a more or less parallel course. Apart from affective changes, inattention, forgetfulness, and fatigability were often noticeable.

Of still more severe mental disturbance instances occurred, in the form of hallucinations, ideas of persecution, etc. Some of the cases of hallucinatory disturbance were very severe, with disorientation in time and space. According to Möbius, the specific chorea psychoses are characterised by hallucinations and disorientation. Seven of the patients exhibited symptoms allied to the motility psychosis (*Motilitätspsychose*) of Wernicke. They made purposeful movements for no obvious reason whatever; one sang, whistled, danced, talked; others made movements of expression; one fastened her quilt round her, beat and struck herself, etc. The behaviour of some was alternately hyperkinetic and akinetic, so to speak. Stereotyped movements, theatrical poses and gestures, monotonous singing, verbigeration, etc., were observed, associated with or followed by negativism, *flexibilitas cerea*, or akinesia in general.

The localisation of choreic movements, inco-ordination, hypotonia, etc., is still a difficult question.

Anton considers the condition explicable by a functional disturbance of the optic thalamus, Bonhöffer of cerebello-thalamic paths. In the view of the latter, the phenomena are due to the absence of centripetal impulses which normally pass by this route to the basal ganglia and the cortex. Clinically, some cases are more cerebellar in type, others tegmental, others thalamic. Many of the symptoms (pain, paræsthesiæ, disturbances of skin, and mucous membrane reflexes, of vessel innervation, sweat secretion, etc.) can be explained by disturbance in basal ganglia and internal capsule. Disease of the former is frequently associated with the occurrence of involuntary movements of expression, such as are seen in chorea. The influence of subcortical disease on the functions of the cortex is important; psycho-sensory symptoms may be due to the arrival of sensory stimuli, impaired and altered in the sensory part of the internal capsule, at the sensory cortex; psychomotor symptoms to the effect of an impaired subcortical co-ordination centre on the motor cortex. But there must in addition, in chorea, be some disturbance of transcortical systems.

S. A. K. WILSON.

A NOTE ON CERTAIN PUPILLARY SIGNS IN CHOREA
(159) F. LANGMEAD, *Lancet*, Jan. 18, 1908, p. 154.

THE following phenomena are stated by Dr Langmead to be of fairly common occurrence in chorea, but to bear no relation to any particular form of that disease, viz., hippus, peculiarities of movement of accommodation, contraction, varying inequality of the pupils, and eccentric pupils. Mere inequality is of little significance in children, but the other signs do not appear to occur in other general conditions except articular or cardiac rheumatism, which is of interest with regard to the etiology of these diseases.

J. H. HARVEY PIRIE.

CERVICAL RIBS AND THEIR RELATION TO ATROPHY OF THE
(160) **INTRINSIC MUSCLES OF THE HAND.** H. LEWIS JONES,
Quarterly Journ. of Med., Jan. 1908.

THE author draws attention to the gradual stages by which a cervical rib has been recognised as a cause of a definite type of muscular atrophy, which chiefly affects the small muscles of the hand.

He describes a series of such cases in St Bartholomew's Hospital Reports for 1893, but the cause of the atrophy was unrecognised.

Buzzard (*Brain*, 1902) described a series of cases under the title uniradicular palsies of the brachial plexus—in some of which at all events a cervical rib was a probable factor. He demonstrated the root distribution of the sensory and muscular affection, but did not recognise a cervical rib as a possible cause of the 1st dorsal root palsy.

Thorburn recognised this association in a paper in which two cases of muscular atrophy of the type in question were described (*Med. Chir. Trans.*, 1905). The author found cervical ribs present in ten out of fourteen cases in which he suspected their presence. He points out that cervical ribs may exist which cause no symptoms. Whether they will do this or not depends on the direction of their growth. He quotes two papers from the *Journal of the Anatomical Society* for 1892 and 1900, in which dissections of the neck were described showing cervical ribs crossed by the 1st dorsal root, and a similar relation has been recognised at operation.

The author refers to a paper by Hinds Howell, with a full report of sixteen cases in the *Lancet*, 1907. Operation on these cases is difficult, but has been successful on several occasions in relief of symptoms.

Reproductions of excellent X-ray photographs illustrating the common types of cervical ribs are included in this paper.

C. M. HINDS HOWELL.

HYSTERICAL PSEUDOTETANY WITH PECULIAR VASOMOTOR

(161) **DISTURBANCES.** (Ueber hysterische Pseudotetanie mit eigenartigen vasomotorischen Störungen.) A. WESTPHAL, *Berl. klin. Woch.*, 9 Dez. 1907, S. 1567.

THIS most interesting article should be read in the original, not only on account of the two important cases there detailed, but for the clearness with which the diagnosis is expounded. In both cases hysterical tetany occurred in relation to peculiar vasomotor disturbances, a connection not previously described. These were local syncope and asphyxia, which, however, never went on to gangrene. Most peculiar were the arterial changes, that exactly resembled those met with in intermittent claudication except that the vessel walls were healthy. Repeatedly in the attacks in one case it was carefully noticed that the pulsation in the dorsalis pedis artery ceased completely.

ERNEST JONES.

CONTRIBUTION TO FREUD'S SEXUAL THEORY OF THE

(162) **NEUROSES.** (Ein Beitrag zur Freudschen Sexualtheorie der Neurosen.) ERNST BLOCH, *Wien. klin. Woch.*, Dez. 26, 1907, S. 1647.

BLOCH relates a case which tends to support Freud's ætiological theory. It concerns a man of 28 who complained of total impotence. Psycho-analysis revealed that when attempting to rape his sister at the age of five his father had caught him and mercilessly punished him. He had forgotten (verdrängt) the incident until the age of thirteen, when he repeatedly failed to perform onanie, each time the picture of his father thrashing him rising to his consciousness and being followed by an end of his erection. Bloch traces his adult impotence to the suppressing action of the painful memory.

Some discursive remarks on Freud's theory are then added. Bloch is convinced of its truth, but not enthusiastic about the practical side of psycho-analysis in therapeutics on account of the extremely great technical difficulties, which he confesses baulk him in ninety-nine cases out of every hundred. He admits, however, the total hopelessness of the older psycho-therapeutics (consolation, reassurance, etc.), and emphasises the superficiality and temporariness of any changes produced thereby.

ERNEST JONES.

ON HYSTERIA AND FREUD'S PSYCHO-ANALYTIC TREAT-

(163) **MENT.** (Ueber Hysterie und die Freudsche psychoanalytische Behandlung derselben.) A. FRIEDLÄNDER (of Hohe Mark), *Monatsschr. f. Psych. u. Neur.*, Bd. 22, Ergänzungsheft, p. 45.

THE author reports briefly seven cases of hysteria treated successfully by him without the employment of Freud's psycho-analytic method, which involves a rather searching investigation of the intimate details of the patient's history. He emphatically dis-countenances such a method in the case of young female patients. One of the patients treated by him had been confined to bed for over twenty years, but recovered completely after treatment lasting over a year.

The author promises a fuller report of the cases, with further therapeutic details.

C. MACFIE CAMPBELL.

GONORRHOEAL SPONDYLITIS. (Spondylose blennorrhagique.)

(164) CROUZON et DOURY, *Bull. et mém. de la Soc. méd des Hôp. de Paris*, 1907, p. 1585.

ALTHOUGH the rôle of gonorrhœa in the production of spondylitis is well established, there are relatively few cases where this ætiology is incontestable. In most of the cases chronic rheumatism, the arthritic diathesis, or tuberculosis can be incriminated. None of these factors can be invoked in the present case. The patient in question had contracted gonorrhœa four years ago. The acute stage lasted three weeks, and was replaced by a persistent gleet. Subsequently pains in the knees developed. A year after the onset of gonorrhœa the patient, who was working in the fields, began to feel pain in the lumbo-sacral region, which spread to the sides of the trunk, the hips, and upper part of the thigh. Simultaneously he developed a stoop. A few weeks before admission he had an attack of gonorrhœal cystitis. On admission the vertebral column showed a considerable curvature, which it was impossible to straighten. Pain in it was spontaneous, and could also be caused by pressure, especially in the lumbo-sacral region. The larger joints were intact. Matutinal gleet was still present.

J. D. ROLLESTON.

POLYARTERITIS ACUTA NODOSA AND PERIARTERITIS

(165) **NODOSA.** W. E. CARNEGIE DICKSON, *Journ. Path. Bact. Camb.*, 1907, Vol. xii., p. 31.

THE writer of this paper endeavours to distinguish between the two above-named conditions. Both are exceedingly rare, only twelve

undoubted cases of the latter and eighteen cases of the former disease having been previously recorded. The author's case of polyarteritis acuta nodosa appears to be the first published in this country, and he therefore deals somewhat fully with the literature of the subject. The case was that of a message boy, æt. 14½ years, whose illness commenced, about a month before admission to hospital, with general weakness, severe epigastric pain, fever and vomiting. Later, convulsions supervened, great emaciation, and ultimately coma and death ten weeks after onset of illness.

Résumé of Post-mortem Examination.—Numerous small, very hard, pale greyish-white nodules, rounded or somewhat spindle-shaped in outline, and varying in size from those just visible to the naked eye up to about the size of small peas, were found on the smaller and medium-sized arteries of the heart, liver, spleen, kidneys, mesentery, brain and spinal cord. The majority of these nodules were about the size of hemp-seeds, and they were evidently situated on or around the arteries, involving their walls, which were at parts distended into minute saccular or spindle-shaped aneurismal dilatations, the majority of which were thrombosed. The latter condition gave rise to numerous small areas of infarction in the lungs and in the kidneys, which also showed evidence of acute or sub-acute inflammatory changes.

The clinical symptom-complex depends largely on interference with the functions of the organs, the vessels of which are selected by the disease. The heart and kidneys are most frequently attacked, and then, in order of frequency, the mesentery and liver, stomach and intestines, muscles, spleen, diaphragm, genital organs, subcutaneous tissue, omentum, peritoneum—more rarely the brain, (*e.g.* in the present case), and, in only one recorded case, the lungs.

General Conclusions.—(1) Under the general term “periarteritis nodosa” two entirely distinct diseases have been hitherto described. These should be differentiated from one another, and classified under different names. (*a*) *Periarteritis nodosa*.—A true periarteritis, nodular in its distribution, the majority, if not all, of the cases of which are syphilitic in nature. (*b*) *Polyarteritis acuta nodosa*.—Characterised by the formation upon the smaller and medium-sized arteries of small localised nodules.

(2) In the case of the latter disease the earliest discoverable changes are found in the outer coat. The process rapidly spreads inwards, so that the most complete destructive lesions produced by the disease are to be found in the muscular coat. These are accompanied by local inflammatory changes, and are followed by the giving way of the internal elastic lamina and the other coats of the vessel wall. Thrombosis of the contents of the lumen and of its aneurismal dilatation is an almost constant accompaniment of the lesion, as are also proliferative changes in the outer and inner coats

of the vessel. Secondary changes, such as infarction, necrosis, hæmorrhage, etc., may occur in the organ or tissue supplied by the affected artery.

(3) The channels by which the infection reaches the vessel wall are possibly the vasa vasorum, or, perhaps, the perivascular lymphatics.

(4) The etiology of the condition is obscure. The cause is almost certainly some bacterial or other infective organism or its toxin. No positive evidence as to the nature of this is afforded by the present case, staining for all the ordinary pathogenic bacteria (including the tubercle bacillus) and for the spirochæte pallida, having given entirely negative results.

AUTHOR'S ABSTRACT.

PSYCHIATRY.

ON THE CLASSIFICATION OF THE PSYCHOSES. (Beitrag zur (166) *Methodik der Statistik und der Klassifikation der Psychosen.*)

TH. ZIEHEN (of Berlin), *Monatsschr. f. Psych. u. Neurol.*, Bd. 22, *Ergänzungsheft*, p. 161.

FOR a satisfactory clinical-symptomatological classification the symptoms of each case of mental disorder have to be studied, not only in their momentary combination and succession, but in their mutual relationship. Each physician has to analyse carefully his own material, to keep to the actual facts of observation, and not to pare down and distort the actual symptoms in order to bring the individual case under a one-word diagnosis. Such a diagnosis is of absolutely no value, and the worthlessness of statistics accumulated on such a basis must be recognised before progress can be made in the classification of the psychoses. Cases should not be forced into antiquated groups, but groups themselves should be formed naturally out of the cases, with full descriptive diagnoses, which do justice to the actual clinical facts. Ziehen illustrates his method of using his clinical material by giving an extract of the entries for four consecutive days in the book containing the record of the diagnoses made. In Ziehen's clinic a provisional diagnosis is entered as soon as the case has been thoroughly examined, and on succeeding visits the diagnosis is reconsidered, and, if necessary, revised. The manner of grouping cases thus analysed is illustrated by giving the results during the first half of 1906. The author does not press the advantages of the classification adopted in his own text-book; he pleads for consideration

of the individual cases unprejudiced by existing descriptions of hard and fast diagnostic groups. In this connection one has to keep in mind the distinction between symptom, syndrome (symptom-complex), clinical picture, and nosological entity.

C. MACFIE CAMPBELL.

A COMPARATIVE STATISTICAL STUDY OF GENERAL PAR-
(167) ALYSIS. CHARLES RICKSHER, M.D., *Amer. Journ. Insan.*,
 Vol. lxiv., Oct. 1907.

THE tabulation of the results of the examination of the histories of the 108 cases of general paralysis received in the Shephard-Pratt Hospital since its opening in 1891. D. K. HENDERSON.

CHANGES IN THE CLINICAL COURSE OF GENERAL PAR-
(168) ALYSIS. (Wandlungen im klinischen Verlaufe der progressiven Paralyse.) R. FELS, *Monatsschr. f. Psych. u. Neur.*, Bd. 22, Ergänzungsheft, p. 34.

THE author went over the case-records of 403 general paralytics to see whether they confirmed Mendel's view that the clinical picture of general paralysis has somewhat changed in the last few decades. The special points to which attention was paid were the social position of the patients, the clinical symptom-picture, the number of paralytic attacks, their nature, period of origin, and relation to clinical symptom-picture, age of onset of the disease; the results are tabulated in a convenient manner.

C. MACFIE CAMPBELL.

CONTRIBUTION TO THE CASE LITERATURE OF THE OCCUR-
(169) RENCE OF PARANOID SYMPTOM-COMPLEXES IN DE-
GENERATES. (Kasuistischer Beitrag zur Lehre von dem Auftreten paranoider Symptomenkomplexe bei Degenerierten.)
 EDUARD REISS (Tübingen), *Centralbl. f. Nervenl. u. Psych.*,
 December 1, 1907.

REISS discusses in this paper a form of acute or sub-acute mental illness which appears in criminal cases, and during sentence, and which generally disappears quickly and surely when the prisoner is released.

This form of mental illness is the first of the three well-defined forms described by Bonhoeffer in his work on the "Psychoses of Degeneration." Siefert has lately given examples of it in his

"Mental Diseases of Prisoners" under the heading "Hallucinatory Paranoiac Form." Neither Bonhoeffer nor Siefert give an explanation of the origin of the disease. They agree in emphasising the fact that it is not due to dementia præcox.

Reiss has seen eight cases. Of this number only three present detailed and complete histories. In all the cases the psychosis had been present for a long time and in most for quite a number of years. The periods of sanity between the outbreaks of illness were from one to eight years' duration. The greater number of cases appeared during imprisonment (generally seclusion). Fear, ideas of reference, delusions of ill-treatment, single hallucinations, irritability and ill-humour, and a tendency to morbid recapitulation of past events are the principal signs. Clearness of mind and orientation remain. Exceptional symptoms in the form of excitement, accompanied by violent outbursts of temper, during which clearness of mind is lost, occur in several cases, but as outbreaks of excitement occurred during the periods of sanity in the several cases, these exceptional symptoms could not be reckoned on as peculiar to the psychosis. The duration of illness was always from a few weeks to several months, and only in one case did it last for over a year. In all cases a complete and unaided recovery was made on the patients being released from the unfavourable surroundings of the prison. The influence of surrounding objects in exciting the psychosis is shown by four of the seven patients suffering a relapse when put into the same conditions as before, and recovering again as soon and as quickly as when released formerly.

The case of an artist, 31 years of age, with a very bad family history in regard to insanity, is gone into in detail. The relationship and diagnosis of the form of insanity in this patient are fully discussed. It is compared with hebephrenia, running an episodic course, true paranoia and manic-depressive insanity, and the conclusion is reached that it is a case of paranoiac illness founded on a basis of degeneration. The history of the patient's illness shows that he was not a criminal. It brings out very clearly the exciting elements of the insanity, viz., outside influences. In the case in question unaccustomed surroundings aroused numerous fears in a timid and mistrustful mind. The mental confusion was aggravated by the repeated and over-anxious warnings of the mother of the patient, who could never emphasise enough the dangers of large towns. With these warnings and with a feeling of irremediable solitude, the patient found himself for the first time in Paris, and felt constrained to leave the city in all haste. The second attack occurred in Madrid, where he quarrelled with the landlord, and the emotional excitement which followed made him an easy prey. The cause of the outbreak in the third instance was very clear. For years he cultivated the idea that he was an anarchist,

and at every opportunity he tried to make himself noticeable by giving vent to revolutionary ideas. Suddenly a strike broke out in his native town, and the arrest of anarchists was a daily topic of conversation. The mother could not warn her son sufficiently against talking of his political ideas, as she knew that he had already made himself noticeable in more ways than one, and, besides this, he was suspected by other people. In this state of mind he arrived in the Balearic Islands, where he had had unpleasantness in the previous year on account of his socialism. For several days his thoughts worried him; then all at once a sharp paranoiac attack of excitement set in which was confined solely to apprehensive conceptions working on his mind, and when he returned home and the disturbing outside influences were removed, he became quite well in a short time. After six months the whole psychosis suddenly returned as a result of his unhappy boasting about his political views. He became excited, quarrelsome, and the former events in the Balearic Islands returned vividly to his mind. Since then the socialistic ideas have been less marked, and during later periods of fear and apprehension he has not had delusions. In this patient there is a peculiar pathological reaction in a degenerate, the form it takes depending on his constitutional peculiarities, its frequency and severity depending for the most part on external circumstances; in many respects, therefore, there was a close relationship to the hysterical psychoses. The psychogenetic phenomena are a widespread property of degeneration. In this psychosis and in all the cases, the complete recoveries made, and the dependence of the affection on outside influences as exciting causes, are noteworthy features.

HAMILTON C. MARR.

A CASE OF DEMENTIA PRÆCOX, CATATONIC FORM (170) (KRAEPELIN), WITH RECOVERY AFTER FIFTEEN YEARS; REMARKS ON THE DIFFERENTIAL DIAGNOSIS. (Ein Fall von Dementia praecox catatonischer Form (Kraepelin) der nach 15. jährigen Dauer in Genesung ausging, nebst differentiell-diagnostischen Bemerkungen.) SCHAEFER (of Roda), *Monatsschr. f. Psych. u. Neur.*, Bd. 22, Ergänzungsheft, p. 72.

THE patient was a well-educated physician of thirty, with a good family history, who developed severe neurasthenia after prolonged overwork; two years later after typhoid fever, and a return of the neurasthenic symptoms, he became acutely insane. At first he showed deep depression, anxiety, weariness of life, suicidal trend; these symptoms were soon followed by a condition of marked confusion with excitement. During the following years various phases

of the disorder were observed; sometimes the condition was one of profound depression with a suicidal tendency, delusions of sinfulness and of having an incurable disease (tabes, etc.), sometimes he was dominated by hallucinations of a depressing nature, sometimes his state was that of catatonic stupor with episodes of violent behaviour.

After many years the symptoms became less marked, and appeared to be succeeded by an apathetic dementia with catatonic traits. After passing through a phase of slight exhilaration the patient, after the uninterrupted duration of the disorder for fourteen years, made a complete recovery. At the time of writing he had for two years been carrying on a large practice, had complete insight into the true nature of his previous disorder, and profound thankfulness for his recovery.

After reporting the case the author makes some pertinent criticisms of Kraepelin's extremely large group of dementia præcox, under which name the latter has brought together various different disease pictures which have in common a termination in a certain form of dementia. Kraepelin himself admits that a certain number of cases do recover—in fact, about 13 per cent. of the cases of this catatonic variety.

In view of this fact, and in face of the case reported, Schaefer considers it not advisable to make a large and somewhat heterogeneous group of which the termination in dementia is to be one of the main characteristics. It would be better to retain the smaller groups and also the conception of secondary dementia for a certain group of cases.

Such a discussion of the question of dementia præcox shows how valuable it would be if Kraepelin or one of his pupils were to publish his dementia præcox material from the Heidelberg Clinic with the histories of the patients up to date.

C. MACFIE CAMPBELL.

PROGNOSIS IN CASES OF MENTAL DISEASE, SHOWING THE

(171) **FEELING OF UNREALITY.** F. H. PACKARD, M.D., *Amer.*

Journ. Insan., Vol. lxiv., Oct. 1907.

THE writer has worked out the practical significance of the symptom "feeling of unreality" in regard to prognosis and diagnosis. Under the head "feeling of unreality" are included all those symptoms arising from the loss of the feeling of reality which Wernicke has divided into the allopsychic, the somatopsychic, and the autopsychic fields. In the allopsychic field such expressions as "The trees seem changed," "People are not real," are common. In the somatopsychic field patients use expressions to the effect that their

stomach or other organs have gone, or that they have no bodies at all, and similarly in the autopsychic field we hear them say, "I am dead," "I cannot die." This symptom was first described by Esquirol, and later, by Cotard who described it almost wholly in connection with cases which Kraepelin called involution melancholia, a condition that had a bad prognosis. Further observation, however, has shown that, although the feeling of unreality occurs very frequently in involution melancholia, it is not an essential symptom, and occurs also in manic-depressive insanity, general paralysis, and some psychoses, which superficially resemble dementia præcox; it has not as yet been recorded, in cases of classical dementia præcox. Formerly cases in which this symptom occurred, were thought to have a bad prognosis, but the writer shows by a number of cases of involution melancholia, manic-depressive insanity, and cases resembling dementia præcox, which recovered or improved, that one may give a fairly good prognosis.

He concludes by saying that the feeling of unreality is not a fundamental symptom, nor a pathognomonic symptom, and not of bad prognostic significance. He emphasises the fact, that the diagnosis and prognosis of cases should be made on the more fundamental symptoms.

D. K. HENDERSON.

DELIRIUM TREMENS. Statistical Study of One Hundred and (172) Fifty-six Cases. J. NAPOLEON BOSTON, *Lancet*, Jan. 4, 1908.

THIS paper gives an analysis of 140 cases of delirium tremens treated at the Philadelphia Hospital (Blocley), between January 1, 1904, and March 1, 1907. There were in all 156 cases treated during this period. But sixteen have been omitted in the analysis on account of incomplete records.

Age was seen to be a predisposing factor to the disease, as well as exercising an influence on its gravity. The greatest number of cases occur between the ages of thirty and fifty, but the death-rate remains almost the same during the second, third, and fourth decades. The prognosis is unfavourable after the age of fifty. Many patients were admitted to hospital for the disease several times: one individual reached a total of forty-two admissions. The popular idea that the disease proves fatal during the second or third attack was not corroborated by the author's experience.

The largest number of cases developed in August; the death-rate during this month was 46·7 per cent., as against the average death-rate 37·1 per cent. December and January showed a death-rate of 72·7 per cent. and 50 per cent. respectively. In spring, when the disease is fairly common, the death-rate is only 7·1 and 10 per cent.

Of twenty-two cases with pre-existing or accompanying complications, ten showed kidney and lung involvement, seven heart and lung, two kidney and heart, three heart and stomach. Thirty-eight cases were uncomplicated, and all of these recovered. All the fifteen cases having renal complications died. Pre-existing cardiac disease gave a mortality of 84.6 per cent., acute cardiac complications being hardly less serious. Cases with bronchitis and bronchopneumonia had a death-rate of 66 per cent.

A. HILL BUCHAN.

ACUTE TRAUMATIC PSYCHOSES. (Zur Kenntnis der akuten (173) traumatischen Psychosen.) M. SOMMER (of Bendorf), *Monatsschr. f. Psych. u. Neur.*, Bd. 22, Ergänzungsheft.

THE report of two cases. The first patient presented Korsakow's symptom-complex, with explosive mood and ideas of greatness, and became definitely demented. The second case was a boy of 13, who, after a severe fall on the occiput, showed complete change of character without any intellectual defect; he was rude, gluttonous, irritable, obscene, and shameless. After a few weeks these morbid symptoms disappeared, and the patient made a complete recovery. The author describes the psychosis as a moral autopsychosis in the sense of Wernicke.

C. MACFIE CAMPBELL.

TREATMENT.

THE FAVOURABLE INFLUENCE OF OCCUPATION IN CER-
(174) **TAIN NERVOUS DISORDERS.** ATWOOD, *N.Y. Med. Journ.*,
Dec. 14, 1907.

THE great value of occupations and diversions in psychotherapeutics is due not only to improvement of general nutrition and metabolism, but to re-education and development of the motor brain centres and increased flow of blood to the parts adjacent to these centres. At first only passive participation on the part of the patient may be available, but this, though of less help than active work, should never be neglected as a means of treatment. Excellent practical results have been obtained in epileptic colonies from outdoor forms of work, suited to the individual requirements and capabilities of the patients. In selecting the occupation suitable for abnormal mental conditions, attention must be paid not only to the actual state of the patient but also to his previous history, so that the work chosen may recall pleasant and not painful or harmful associations. The effects of music depend more on the instrument employed than on

the nature of the musical production ; thus, the soothing strings and horns, the stirring wind instruments of wood, the even more enlivening brass instruments, and the agitating and thrilling drum and piccolo, have each their own peculiar place in treatment, and are not to be indiscriminately exhibited to all types of disease. As regards amusements, games of various kinds (especially golf, the dosage of which is easily regulated), calisthenic drills, whist, dancing, and even the study of geography, arithmetic, etc., assist in breaking up the continuity of morbid ideas and mischievous acts. Handicrafts, such as clay modelling, cabinet-making, and wood-carving, preceded, if necessary, by a period of complete rest, hasten the restoration to health and self-confidence. Such methods of treatment are most easily carried out in hospitals, but in private practice the adoption by the patient of some fad has frequently a most beneficial effect.

HENRY J. DUNBAR.

**THE CURABILITY OF A RARE FORM OF NOCTURNAL PETIT
(175) MAL BY THE USE OF LARGE DOSES OF BROMIDE.**

L. PIERCE CLARK, *Amer. Journ. of Med. Sc.*, Jan. 1908, p. 94.

IN this rare form of epilepsy, which has often been stated to be a form of hysteria, the attacks invariably occur while the patient sleeps, usually beginning as soon as the patient falls asleep. There may be as many as three hundred separate attacks per night. The patients usually awaken from deep sleep. The eyes open widely, while the pupils are dilated and unresponsive to light. The face is either very congested or very pale. In a few seconds the patient executes some incredibly rapid movements of the hands or feet, movements without any intent or purpose. Then, unless disturbed, he soon passes into normal sleep again.

In this type of case bromides in ordinary doses increase the frequency and severity of the attacks, but the writer here records four cases which he has treated with large doses (130-400 grs. daily) in which there has been immunity from paroxysms for several years. Great care and attention are needed during the period of what is, really, bromide poisoning; the details of the treatment are given.

J. H. HARVEY PIRIE.

THE TREATMENT OF TRIGEMINAL NEURALGIA BY INJECTIONS OF ALCOHOL. BRISSAUD and SICARD, *Rev. neurologique*,
(176) Nov. 30, 1907, p. 1157.

THIRTY-THREE cases of right trigeminal neuralgia, and eleven of left, were treated by injections of alcohol. A fine needle, 4, 5, or

6 centimetres long, with a diameter of seven-tenths of a millimetre, is employed. The strength of the alcohol used is 80 per cent., and the amount injected is a half or one cubic centimetre. Reference must be made to the original for the description of the various foramina of exit of branches of the trigeminal and its divisions, and how to reach them with the needle. Stovaine is used by the authors as a local anæsthetic, and the injection is always made with the patient in the horizontal position.

The pain of the injection usually passes off after a short time, sometimes almost immediately. Sometimes the part treated becomes œdematous for a time. If the injection is successful the area of distribution of the branch concerned ought to be anæsthetic. This anæsthesia persists, as a rule, from four to eight months, sometimes much longer. Sometimes there is myosis, if the sympathetic filaments of Hæckel's ganglion are reached by the alcohol. Paræsthesiæ in the anæsthetic area are not infrequent.

The authors have had excellent results in their cases, some of which have been followed for as long as eighteen months. Any return of pain after a time has been treated successfully with reinjection.

They recommend this form of treatment not merely for idiopathic trigeminal neuralgia, but for any secondary facial neuralgia.

S. A. K. WILSON.

THE INDICATION FOR TENDON OPERATIONS IN SPINAL AND (177) CEREBRAL PALSIES. (*Die Indikation zu Sehnenoperationen bei spinalen und cerebralen Lähmungen.*) P. BADE (of Hanover), *Wien. med. Wochenschr.*, Nov. 9, 1907.

THE author defines more precisely than has previously been done the indications for tendon operations in the above conditions. It is inadvisable to operate until two years after the onset of the palsy, as even in the second year there may be some regeneration of muscle; extensive tendon operations should not be carried out in children under six, owing to the nature of the tendinous structures, the difficulty of putting up the limb in good position, and of carrying out satisfactorily the after-treatment. Operations in Little's disease may with advantage be carried out after the third year.

Tendon operations are only indicated when the muscular equilibrium is considerably disturbed; the greater the contracture and the deformity, the clearer is the indication for the operation. The aim of the operation is not to give the limb merely its normal form, but to restore to it approximately normal function. The author refers in more detail to a few definite deformities.

C. MACFIE CAMPBELL.

ON SPINAL ANÆSTHESIA. (Ueber Rückenmarksanästhesierung.)(178) A. REMENAR, *Wien. klin. Wochenschr.*, Nov. 7, 1907.

THE author gives the results of his use of spinal anæsthesia in surgical operations, the various drugs employed being stovaine (40 cases), tropacocaine (36 cases), novocaine (4 cases). The more poisonous the preparation, the more complete the anæsthesia. With novocaine the anæsthesia is complete, the motor paralysis almost complete; with tropacocaine some sensibility may remain, the patient occasionally feels a dull pressure but no pain; stovaine was given up, as cases had been reported with nuclear pareses, myelitis, death due to respiratory paralysis. The author in only one of the cases where stovaine was used saw slight collapse.

His method is to inject 1 c.cm. of a 4 per cent. solution of tropacocaine in pure water into the spinal canal. The puncture is made with the patient in the sitting posture, leaning well forward; by means of the Trendelenburg position the anæsthesia may be made to reach even the costal margin. Bier, by making the patient take deep inspirations with mouth and nostrils closed, was able to anæsthetise the whole trunk, axilla, and upper extremities.

In two of the thirty-six cases treated with tropacocaine anæsthesia was incomplete, but narcosis was not necessary; in two other cases slight narcosis was necessary.

The operations included Whitehead's operation for hæmorrhoids, resection of the knee and of the ankle joint, amputation of the leg. The age of the patient is no contra-indication, the method having been used with children of ten and patients of eighty. The merits and contra-indications of the method are discussed.

C. MACFIE CAMPBELL.

THE TREATMENT OF CHOREA MINOR, WITH ESPECIAL(179) **REFERENCE TO THE DANGERS OF THE ARSENIC THERAPY.** KOPLIK, *Med. Rec.*, 18th Jan. 1908.

ALTHOUGH chorea has come to be recognised as an infection pure and simple, the therapy of the disease has, generally speaking, not been altered to meet the indications of improved pathological knowledge. The application of routine empirical treatment to all forms and degrees of chorea is especially irrational, cases with associated endocarditis or paralytic phenomena requiring entirely different hygienic and medicinal measures from those free from such complications. The administration of arsenic is, in the writer's opinion, exceedingly dangerous in this disease in the large doses in which it is usually administered. The commonly recognised symptoms of arsenical poisoning, such as neuritis, gastric disturbance, cedema, and skin eruptions, are by no means infrequent; but the toxic action on the kidneys is a much earlier

and more constant symptom of poisoning. As a result of careful investigation in a series of cases, the writer has formed the opinion that the appearance of albumin, casts, and even blood in the urine is a frequent phenomenon during the treatment of chorea by arsenic, and that the examination of the urine gives the most delicate test for the point of tolerance of the drug. Sunshine, fresh air, gentle recreation, and the society of other children are beneficial in ordinary mild cases. When endocarditis is present as a complication the treatment will have to be modified so as to secure almost complete rest, and in the severer types with paralytic symptoms isolation will be indicated. Arsenic should only be given in the simple cases, and then only in tonic doses. In cases with mental instability, or with cardiac involvement, strychnine as a bitter tonic is very useful, and hydrotherapy is frequently of great service. Trional, chlorotone, and the bromides, all in small doses, are the most suitable sedatives to employ should occasion arise.

HENRY J. DUNBAR.

LUMBAR PUNCTURE IN OPTIC NEURITIS. STEPHENSON, *Med.*
(180) *Press and Circ.*, 12th Feb. 1908.

THIS is a summary of a paper with reports of cases by Babinski and Chaillons. An abstract of the leading facts of seven cases where improvement followed the operation is given. These writers "conclude that lumbar puncture may be considered as a curative method in optic neuritis due to intra-cranial effusion. On the other hand, it is merely palliative in neuritis due to intra-cranial tumour. The operation, under any circumstances, should be performed with care, and the amount of fluid evacuated should be in inverse proportion to the severity of the symptoms of compression. It is better to renew the puncture as often as necessary rather than to do too much at one time."

HENRY J. DUNBAR.

Review.

**LE ALTERAZIONI DEL SANGUE IN RAPPORTO SPECIALMENTE
ALLE MALATTIE MENTALI.** RAFFAELE GALDI, *Nocera
Inferiore*, 1907.

THIS work is an interesting and important compilation of the results which have been obtained by a large series of investigations into the changes in the blood in the various insanities.

In the earlier part of the work the coagulability, density, and alkalinity of the blood, and the qualitative and quantitative changes of the various blood corpuscles, are dealt with.

It is evident, from the divergent and even contradictory results obtained, that the subject is one of considerable difficulty, and that the methods of investigation must be improved, and more uniformity of technique established, before we can expect much reliable information with regard to these changes. It must be remembered, also, that the connection between the condition of the blood and the mental disturbance is often an indirect one, and that there are various factors which may be active in some cases and not in others.

More interesting is the second portion of the work, which deals with the hæmolytic and bactericidal power of the blood, with serum therapy in some mental diseases, and with the presence of bacteria in the blood in cases of acute delirious mania, dementia præcox, epilepsy, and general paralysis. But in all these branches of the subject the discrepancies are too marked to allow of any definite statement.

The chapter on serum therapy is devoted chiefly to an account of the serum inoculations of epileptics by Ceni.

With regard to the finding of bacteria in the blood of the insane, the author is of the opinion that although several varieties of micro-organisms have been isolated from the blood in different forms of insanity, it is not yet settled whether these can be considered as the cause of the insanity, or whether they have invaded the tissues during the last stages of life. He does not admit that a specific organism has yet been found for any mental disease.

Such a work as this was wanted, and Dr Galdi has rendered a great service by bringing together in what he calls a "Synthetic Review" the results which have been so far obtained in this important branch of the subject of insanity.

R. G. Rows.

BOOKS AND PAMPHLETS RECEIVED.

Georg Lomer. "Bismarck im Lichte der Naturwissenschaft." Carl Marhold, Halle, 1907, M. 3.

Paul Guerrier. "Étude Médico-Psychologique sur Thomas de Quincey." A. Rey, Lyon, 1908.

A. Hoche. "Moderne Analyse psychischer Erscheinungen." Fischer, Jena, 1907, M. —50.

Paul Kronthal. "Über den Seelenbegriff." Fischer, Jena, 1905, M. —50.

Ivar Wickman. "Beiträge zur Kenntnis der Heine-Medinschen Krankheit (Poliomyelitis acuta und verwandter Erkrankungen)." Karger, Berlin, 1907, M. 6.

Merzbach. "Zur Psychologie des Falles Moltke." Hölder, Wien, 1908.

"Psychiatrie Contemporaine," No. 1, 1908. Soukhanoff, Moscow.

Review **of** **Neurology and Psychiatry**

Original Articles

THE CYTOLOGICAL STUDY OF THE CEREBRO-SPINAL FLUID BY ALZHEIMER'S METHOD, AND ITS DIAGNOSTIC VALUE IN PSYCHIATRY.¹

By HENRY A. COTTON, M.D.,

Medical Director, N.J. State Hospital at Trenton ; formerly of Danvers
Insane Hospital, Hathorne, Mass. ; and

J. B. AYER, Jr., M.D.,

Assistant Pathologist, Danvers Insane Hospital.

METHODS.

THE value of lumbar puncture as a diagnostic aid in psychiatry and neurology has been nullified to some extent by the defects of technique rather than by a lack of specific changes in the fluid due to pathological causes.

Prior to the year 1904, the centrifuge method of Widal and Ravaut was practically the only method for studying the cytology of the cerebro-spinal fluid. It answered a certain purpose very well, but the inaccuracies were soon apparent ; and while clinicians made use of the method for diagnosing general paralysis, the pathologists generally severely criticised it, and doubted if it had any diagnostic value.

In the first place the cells were so very poorly stained that one

¹ Read at the semi-annual meeting of the New England Society of Psychiatry at the Colony of the Worcester Insane Asylum (Grafton, Mass.), Sept. 19, 1907.

could not distinguish the various types, and the only value was in finding an increase in the number of nuclei seen, without any regard to what cells were present to account for the increase. The cells were usually spoken of as lymphocytes, and the increase called a lymphocytosis. It is true that in certain cases of general paralysis there is a great increase in the number of cells, but the protean character of the disease, its extent and intensity, allows of many anatomical types. Hence we should look for some pronounced variation in the number and character of cells found in the cerebrospinal fluid. It is often difficult, by the above method, with a small count of cells, to decide whether to call the results positive or negative. Nissl (1) called attention to the fact that by this method a large count per field—8 or 10 cells—could occur in fluids without being of any pathological significance. The difficulty of comparing the results of various observers was also apparent, as one must necessarily perfect one's own technique and draw conclusions from experience—establishing one's own standards. Pomeroy (2) uses this method and reveals the inaccuracies of the same when he concludes that all the clinical facts must be considered before an opinion as to the cell count can be given. He also states that it is impossible to differentiate various cell types because of their poor staining qualities. It is very easy to see that in a clinically well-marked case of general paralysis lumbar puncture is superfluous as a means of diagnosis. But in very doubtful cases, where the physical signs are either absent or not sufficiently pronounced to warrant a diagnosis of general paralysis, lumbar puncture should be of the utmost value in aiding us to arrive at a positive diagnosis.

Fuchs and Rosenthal (3), in 1904, in order to overcome the inaccuracies of the centrifuge method, and establish some standard for comparison, utilised the ordinary hematological technique, the pipette and blood-counting chamber. The fluid was not centrifuged, but drawn directly into the pipette, and a diluent used that would stain the cells. Knowing the degree of dilution, one could count the cells present in so many c.c. of fluid. Here at last a constant unit could be employed, and with a large number of cells per c.c., the method was fairly accurate. But with a small number of cells, the errors would vary from 30 to 90 per cent.

Jones (4) has shown how the inaccuracies could occur in counting a small number of cells. He proposes another method of utilising

the same principle, but we fail to see the advantages of his method over that of Fuchs and Rosenthal.

Cornell (5) utilises the last-mentioned method and obtains some good results. It must be admitted that the method is far in advance of the centrifuge method of old, as it allows to some extent a differential count (just how accurate will be discussed later). Cornell was able to differentiate the following cells :—

- | | |
|------------------------|-----------------------|
| 1. Small lymphocytes. | 4. Epithelioid. |
| 2. Large lymphocytes. | 5. Plasma cells. |
| 3. Polymorphonuclears. | 6. Degenerated cells. |

The lack of illustrations is a serious defect of this work, as one is in doubt as to the character of the cells from the description alone. This is especially true of the epithelioid cells. He was able to distinguish plasma cells, which were first described in the fluid by Fischer; the latter's results, however, were doubted by Nissl and others, because they were unable to distinguish these cells. One serious objection to the method is the fact that the cells are not fixed in the usual manner, and that in staining the fresh cells, they appear necessarily distorted and swollen. The inability to compare these cells with cells in the tissue, fixed and stained by the common methods, is a serious obstacle in accounting for their origin. We will also show later that Cornell failed to observe other cells that are of considerable importance in diagnosis. The lack of anatomical confirmation of his diagnosis is also to be regretted, as we cannot always be satisfied with the diagnosis of general paralysis made from the clinical picture alone.

Realising the importance of lumbar puncture, and the defects of the methods in use, Alzheimer (6), after much experimentation, finally evolved a method by which the cells are fixed with alcohol, after which they can be stained by the usual methods used in studying the histopathology of the cortex. This is accomplished by adding 96 per cent. alcohol to the cerebro-spinal fluid, which precipitates the proteid, and by centrifugalisation the cells are thrown down with the proteid in the form of a coagulum at the bottom of the tube. By his method a very clear differentiation of the various types of cells can be made, due to the excellent staining qualities of the cells thus treated.

In detail, the method of Alzheimer as used by us is as follows :—

1. Lumbar puncture in the usual manner.

2. 96 per cent. alcohol, in proportion to twice the amount of cerebro-spinal fluid, is added drop by drop and well mixed.
3. Centrifuge the mixture for one hour at high speed in a glass tube with conical end. (An ordinary electric urinary centrifuge apparatus can be employed, tube to be well stoppered to prevent evaporation.)
4. The supernatant fluid is poured off, leaving a small coagulum in the bottom of the tube.
5. Add absolute alcohol—alcohol and ether—ether, each separately for one hour, to dehydrate and harden coagulum.
6. The coagulum can now be gently loosened from the bottom of the tube by a long needle. The tube is then inverted, and the coagulum allowed to fall into the hand by a quick tap on the end of the tube. Care must be taken not to squeeze or handle the coagulum. The hand is placed over a small homeopathic vial, containing thin celloidin, and the coagulum allowed to drop into the celloidin, where it remains over night (12 hours usually).
7. Coagulum placed in thick celloidin, which is allowed to evaporate slowly.
8. Then mounted on blocks, and cut at 14 m.
9. Sections stained. (Celloidin should be removed from section by alcohol and ether before staining.)

The stains used by Alzheimer and by us were Unna's polychrome methylene blue and Pappenheim's pyronin-methyl green.¹ The

¹ *Pyronin Stain*—

Methyl green	0.30.
Pyronin,	0.25.
Alcohol, 96%.	2.50.
Glycerin	20.
5% aqueous sol. carbolic acid,	100.

Procedure—

1. Remove celloidin by abs. alc. and ether,
2. 80% alc.
3. Water.
4. Sections are carried on glass or platinum needle into dish of above sol. kept in a water bath at 40° C., 5-7 mins.
5. Quickly cool dish in running water.
6. Wash all superfluous stain in plain water.
7. Absolute alcohol to differentiate—until no more stain comes away from section.
8. Clear in Bergamot oil.
9. Mount in balsam.

CYTOLOGICAL STUDY OF CEREBRO-SPINAL FLUID 211

latter was found to be the most satisfactory routine stain, as it gives excellent nuclear pictures, a slight tint to the protoplasm in most cells, and is considered specific for plasma cells, staining the protoplasm a deep red. Toluidin blue was also used with success when especially clear nuclear figures were desired; Scharlach R. was used to demonstrate the fat found in "Körnchen" cells. Instead of celloidin imbedding, paraffin may be used, with alcohol or Zenker fixation. This was tried by us, but the results did not seem to be as satisfactory as with the technique outlined above, the only advantage of the method being that sections could be cut a little thinner.

As to the method described by Alzheimer, we can say that it is the most satisfactory one yet devised. Because of the fixation, cells are stained in a manner easy of differentiation, and these cells can be compared with cells in the pia and cortex (stained in a similar manner), an interesting point when the origin of the cells has to be considered. The contrast between this method of treating the cerebro-spinal fluid and others in vogue before its publication can only be appreciated by one who has attempted to overcome the difficulties and inaccuracies of the latter, and to Alzheimer belongs the credit of devising a method that allows an accurate study of the cytology of the cerebro-spinal fluid in normal and morbid conditions.

The cells are caught in the coagulum and are found to be nearly evenly distributed throughout (see Fig. 6, Plate 16). It is possible that the very topmost layers of the coagulum may not contain as many cells as the bottom layer, but this is only true when a very small number of cells are present. But by cutting the coagulum in cross section, and staining at least six sections from various levels, and averaging the counts from these various sections, very little error as to character and number of cells is made. While the method is not a bedside one and requires some little time, yet the advantages are so great that one is amply repaid for the extra time spent upon the procedure. When the cells were present in sufficient numbers a differential count was made from 200 cells, using about six sections for the purpose. The unit for comparison of the counts in various fluids was the number found in 100 fields, as it was found that, in conditions other than general paralysis, the cells were so scarce it would be ridiculous to speak in fractions of a cell to one field, and the error would be greater. On the other hand, in general paralysis the number becomes large, up to 3400 cells in some cases

to 100 fields, but it is only necessary to count 200 cells, and keep track of the number of fields counted, and by simple multiplication to get the totals for 100 fields. Here the differential count is of the utmost importance.

MATERIAL AND SCOPE OF INVESTIGATION.

We have been fortunate enough to have at our disposal a large amount of material, both living and post mortem, and thus we have been able to study the cerebro-spinal fluid in both conditions; also to confirm our diagnosis and correlate our findings in the fluid by studying the cortex and pia in a number of cases. This correlation of the findings in the fluid with the cortical histopathology has been of great value, not only in confirming the clinical diagnosis, but also in establishing the identity of the various cell types.

Our cases total 82, and in some instances two punctures during life were made. In 3 cases of general paralysis, and 3 cases of organic dementia, both ante-mortem and post-mortem punctures were made, and in 2 cases of each of the above series autopsies were made. A detailed summary of all fluids will be found in Chart I. The number of fluids in various psychoses are as follows :—

- 19 cases General Paralysis, A.M.
- 12 „ General Paralysis, P.M. (Three punctured A.M., and autopsies in two of these, and also in five other cases.)
- 10 „ Organic Dementia (arteriosclerotic, etc.), A.M.
- 8 „ Organic Dementia. (Three punctured A.M. Autopsies in five cases, including two punctured A.M. and P.M.)
- 3 „ Senile Dementia, A.M.
- 3 „ Senile Dementia, P.M.
- 2 „ Polyneuritic Delirium, A.M.
- 1 „ Chronic Alcoholic Insanity, A.M.
- 1 „ Chronic Alcoholic Insanity, P.M.
- 4 „ Epilepsy, A.M.
- 4 „ Manic-depressive Insanity, A.M.
- 1 „ Manic-depressive Insanity, P.M.
- 1 „ Involution Melancholia, A.M.
- 9 „ Dementia Præcox, A.M.
- 1 „ Dementia Præcox, P.M.
- 1 „ Paralysis Agitans, P.M.
- 1 „ Paranoia (?), P.M.

CYTOLOGICAL STUDY OF CEREBRO-SPINAL FLUID 213

2 cases Idiocy (Spastic paraplegia) P.M. Died of cerebral softening.

1 „ Morphinism, P.M.

1 „ Toxic Delirium, P.M.

1 „ Cerebral Lues, A.M.

1 „ Neurasthenia, A.M.

Doubtful Cases.

2 „ Organic Dementia (?) (1 A.M., 1 P.M. no autopsy).

2 „ General Paralysis (?) (1 A.M., 1 P.M. no autopsy).

The primary object of the investigation was to compare the fluid of various psychoses, especially those psychoses that would be confused with general paralysis clinically, and we believe that by this method facts have been obtained that warrant the statement that changes occur in the cells of the cerebro-spinal fluid that are pathognomonic of general paralysis, so that from the fluid alone a positive diagnosis should usually be possible. (Of course this refers only to patients suffering from mental diseases.)

In order to substantiate our claims, we have utilised the anatomical material at our disposal, both to confirm the clinical diagnosis and establish the origin of the various cells. We have not taken up other conditions outside of the realm of psychiatry from lack of time and material, so that our conclusions refer entirely to this field.

In such conditions as organic dementia, dementia præcox, alcoholic insanity (acute and chronic), senile dementia and epilepsy, we have compared the findings with those of general paralysis and have come to definite conclusions. Many interesting points relating to the changes in post-mortem fluids have arisen, but we will only be able to mention them and their relation to acute toxic conditions, and we hope that, aside from obtaining facts for diagnostic purposes, this work will stimulate investigation in other fields of medicine, especially the post-mortem fluids in general diseases.

CYTOLOGY.

As we have stated above, no method previously in vogue has allowed such a perfect differentiation of the cells of the cerebro-spinal fluid. Consequently, we not only have been able to distinguish cells found by other observers, but have found cells

that we believe have not been described previously in the cerebro-spinal fluid.

It is no doubt true that certain cells have been incorrectly classified before, because of poor staining qualities due to the methods used. We can be reasonably sure of the identity of lymphocytes, plasma cells, endothelial cells, phagocytes (endothelial class with lymphocytic inclusions), polymorphonuclear leucocytes, and "Körnchen" cells. Other cells are found in small numbers, and have been put in the unclassified list. They may be degenerated types of cells already mentioned. Some cells resemble fibroblasts and ependymal cells, but it is difficult to come to any definite conclusions as to their identity.

Nissl doubted if the cells of the cerebro-spinal fluid could come from the pia, because he found plasma cells in the pia in general paralysis, but was unable to demonstrate these cells in the fluid of patients with this disease, an error doubtless due to the technique employed. Alzheimer, in describing his method, mentions that plasma cells can be clearly seen by this method, and we can confirm his statements. It is also difficult at times to distinguish between large mononuclear leucocytes and endothelial cells in the fluid, but the presence of the former in a fluid with practically no polymorphonuclear cells would not be in harmony with our knowledge of the relation of mononuclears to the polymorphonuclears in the blood. And as contamination of the fluid with blood is in a large measure responsible for any excess of polymorphonuclears, the identity of such cells can be easily established. In most cases, then, where polymorphonuclear cells are absent, mononuclear cells, not lymphocytes, are better classed as endothelial.

1. LYMPHOCYTES.

(*Fig. 1, Plate 17.*) (*A, Fig. 1, Plate 15.*)

We include both the large and small forms as differentiated by Cornell, also altered and transitional forms. The ordinary lymphocytes are found in all fluids, but aside from fluids of general paralysis they occur in very small numbers. The nucleus is small and round; sometimes oval, and slightly indented. The chromophilic granules are arranged in "clock-face" form around the periphery, and take a deep blue stain (pyronin stain). The protoplasm in unaltered forms is found as a thin line around the nucleus, and stains a faint

pink. It is usually wider on one side. The altered and transitional forms show a somewhat larger and deeper staining nucleus, and more protoplasm (Fig. 10, Plate 17). These altered forms are very common in general paralysis, and it is in harmony with the view regarding the origin of plasma cells to consider these altered forms as transitional states between lymphocytes and plasma cells. The lymphocytes come from the pia, and their excess in the fluids of general paralysis is easily explained by the fact that in this disease the pia is infiltrated by them in large numbers, especially in the adventitial sheaths of the blood vessels.

In general paralysis the differential count shows that they are the principal cells that are increased, varying from 33 to 94 per cent., and averaging 73 per cent. of the total count. The total count in general paralysis averages 450 to 1000 fields, so that it is easy to see the actual and relative increase in the number of lymphocytes in this condition. In other conditions the total number present does not reach nearly the count in general paralysis. In cerebral arteriosclerosis (organic dementia) the average is only 23 to 100 fields, so that it is hardly possible that the two conditions would be confused from the lymphocyte count. The lymphocyte count is small in all other conditions examined, being highest in cerebral lues, *i.e.* 36 to 100 fields (see Chart II.). In dementia præcox, the average lymphocyte count is larger than in manic-depressive insanity, but the difference is slight, and at present must be left unexplained, as not enough cases have been examined to allow us to come to definite conclusions. In post-mortem fluids, generally, there is an actual increase in the number of lymphocytes, especially in general paralysis and cerebral arteriosclerosis, but in the former the percentage is lowered from 73 to 62.

2. ENDOTHELIAL CELLS.

(Figs. 4 and 5, Plate 17.) (B, Fig. 1, Plate 15.)

These cells are also a constant finding in all fluids examined (except one case of neurasthenia), but vary in number in the different diseases. They vary considerably in size and shape, often they are the largest cells found in the fluid. The nucleus is usually eccentrically placed, and is oval or "horse-shoe" in shape according to the various stages of its activity. The nucleus stains a faint blue

with pyronin stain and has very few chromatophilic granules. The protoplasm stains a homogeneous light pink and varies in amount. These cells, even when very small, are easily distinguished from lymphocytes, because of lack of chromatophilic granules and shape of nucleus: as they appear in so many forms they may be taken for new types of cells or resemble other familiar cells. Under certain pathological conditions they become phagocytic for lymphocytes and occasionally for their own type. They undoubtedly come from the lymph spaces in the pia, and are easily affected by pathological conditions. The average count of these cells in general paralysis is only 13 per cent. in the living fluid, but they increase to 24 per cent. in post-mortem fluids. They are only present 28 to 100 fields in organic dementia during life, but post-mortem fluids show an increase of these cells to 68 per cent. in this disease. As they show an increase post-mortem in other conditions where small numbers were found during life, it may be possible that the acute diseases which are the cause of death in these conditions may be responsible for this increase. Especially is this seen in organic dementia. In other cases where the post-mortem fluids have shown a relative and actual increase in these cells, the patients have died of some acute disease. This may be accounted for by the fact that at the time of death some changes occur which allow or cause desquamation of these cells so that they appear in large numbers in the fluid. There is apparently no relation between the length of time post-mortem fluid is taken and the number of the cells found, as in the case that showed the largest number the fluid was withdrawn one half-hour post-mortem. The reason for this increase will have to be left in doubt at present, as sufficient proof is not at hand for conclusive statements.

3. PHAGOCYTES.

(*Figs. 1, 2, 3, Plate 17.*) (*Fig. 2, Plate 15.*)

Under certain pathological conditions endothelial cells become phagocytic. They have been described by Mallory in certain toxic conditions, such as typhoid fever. Here, of course, they are found in the tissues. They have also been described as occurring in the pia in epidemic cerebro-spinal meningitis (7), lately by Stuart M'Donald (8), and in tubercular meningitis they are numerous. But we have not as yet seen any description of these cells as occurring

in the cerebro-spinal fluid. M'Donald describes these cells and shows drawings of the same in the pia, but says nothing about their occurrence in the fluid. In cerebro-spinal meningitis these endothelial cells become phagocytic for polymorphonuclear leucocytes. Those found by us are phagocytic chiefly for lymphocytes. This difference in phagocytosis is readily harmonized when the acuteness and chronicity of the processes in which phagocytosis in each case occurs is considered. The nucleus is pushed towards the periphery, somewhat elongated and flattened, and the protoplasm is swelled to enormous proportions.

The lymphocyte is centrally placed and surrounded by a light area or court. The outline of the protoplasm is only seen as a faint line outside of the lighter court. Often the lymphocytes are undergoing degeneration or digestion by the phagocyte, and consequently present various forms of karyorrhexis.

In some cases we have seen these phagocytes with endothelial inclusions, but it is difficult to distinguish between these types and degenerated lymphocytes (see Plate 17, Fig. 2).

Phagocytes were found in the fluid in very small numbers in four cases (living) in a series of nineteen general paralytic fluids, but were found in eight out of twelve cases of general paralysis, and in larger numbers, post-mortem. They are found in the pia in cases of general paralysis and organic dementia, but in small numbers. They evidently become phagocytic *in situ*. From the fact that they are so numerous post-mortem, one would infer that it was entirely a post-mortem phenomenon, but their occurrence during life is against this view. And as in the case of the endothelial cells, the length of time post-mortem seems to have no influence on their quantitative occurrence. The cause of death seems to have no relation to the number found.

4. PLASMA CELLS.

(Figs. 6, 7, 8, 9, Plate 17.) (Fig. 3, Plate 15.)

These cells have a nucleus similar to that of the lymphocytes, except that in most cases the clock-like arrangement of the chromophilic granules is more pronounced; the remainder of the nucleus is stained a deeper blue. The nucleus is oval or round, and eccentrically placed. Frequently plasma cells are found with two or more nuclei, and are considered as degenerate types, the same as de-

scribed by Alzheimer in the cortex of general paralysis. Fig. 4, Plate 15, and Fig. 12, Plate 17, show a cell with a mitotic figure, whether a plasma cell or not cannot be determined. These mitotic figures are frequently seen in fluids of general paralysis. The protoplasm, by the pyronin, stains a deep red or pink (according to the extent of differentiation), and as the pyronin stain is supposed to be specific for the cells, it can be seen how important this stain is for the purpose of differentiation. Around the nucleus is usually seen a lighter area with the protoplasm on the periphery deeply stained. The protoplasm varies in form presumably according to the stage of cell-growth, and many young forms are hard to differentiate from lymphocytes. They differ but little from the cells found in the cortex and pia of brain and cord. So far in our series we have found them only in paralytic dementia, and have found them in all of these cases. Cornell found them in twenty-seven out of thirty-two cases, presumably in general paralysis, but does not mention their significance from a diagnostic standpoint. He also finds them in from 0.1 to 15 per cent., averaging 1.5 per cent. We were unable to find such a high percentage of plasma cells in general paralysis in our series; they vary from 1 per cent. to 6 per cent., and average 2 per cent., or, better described, one seldom sees over one or two plasma cells in a whole section. At present we believe that they are pathognomonic of general paralysis and are of equal value as a diagnostic factor with lymphocytosis. In one of our cases confirmed by autopsy (No. 26), there were only 110 cells seen in 100 fields, and 94 per cent. of these were lymphocytes and two plasma cells. When the fluid was obtained after death the plasma cells had increased to 8 per cent. The diagnosis of general paralysis was not only confirmed by a study of the cortex, but plasma cells were found in abundance in the pia of the cortex and cord. In all the cases of fluid taken P.M., there is a decided increase in plasma cells, varying in twelve cases from two to nine, averaging 5 per cent., a 3 per cent. increase over the cells during life. As to their occurrence in other syphilitic conditions, we have had but one case diagnosed as cerebral lues, and in this case they were absent. In two cases of organic dementia, post-mortem cells resembling plasma cells were found in the fluid, but only one seen in each case, and their identity was questionable. As they were not found in the pia of these cases, their presence in the fluid, if these are classed as plasma cells, cannot be explained.

5. "KÖRNCHE" CELLS.

(Fig. 13, Plate 17.) (Fig. 5, Plate 16.)

This is another type of phagocyte cell, filled with numerous fat droplets or fatty pigment. They were not found in the fluid of any living case. In one case of arteriosclerotic brain disease (No. 65), with a focal softening in the first temporal convolution, the fluid taken post-mortem from the ventricles (lumbar puncture was unsatisfactory) showed a large number of these cells. As the brain had been handled, it may be possible that the softened area was damaged so that these cells escaped. The finding of such cells in the fluid is important from the fact that they can be identified and may help to locate the softening that has broken into the ventricle. One "Körnchen" cell was found in the case of an idiot (No. 83) (also ventricular fluid), who was subsequently found to have extensive cerebral softening. Hence we shall give them some attention. By pyronin, the nuclei of the cells show up darkly stained, about the size of a lymphocyte nucleus. It is eccentrically placed, and the protoplasm is bulged out, usually round or oval, the fat droplets taking a variety of shades of brown. With Scharlach R. they can easily be identified, as they stain a dark red, and although the material is hardly suitable for such a stain, still these granules can be definitely identified as fat. These cells were found in the pia in both cases of arteriosclerotic brain disease.

6. POLYMORPHONUCLEAR LEUCOCYTES.

(Fig. 15, Plate 17.)

From the observation of others, these cells have been given special importance when found in the fluid of general paralytics. Cornell lays special stress on increase of polymorphonuclear cells after epileptiform seizures in general paralysis. With the pyronin stain, the nuclei only are stained. No protoplasm is visible, and they are easily differentiated.

We found that they occurred in nearly all of our cases of general paralysis, varying from 1 to 39 per cent., even in clear fluids, and that they were present also in other conditions (also in clear fluids), but in very small numbers. Wherever we have found these cells in large numbers, however, it has been, with few exceptions, in fluids that were contaminated with blood at time of puncture.

In two cases of general paralysis (Nos. 22 and 71), irrespective of

seizures, the count was 30 per cent. and 39 per cent. respectively, but it was distinctly noted at the time that the fluid was "bloody." In one case (No. 35), however, without seizures and apparently a clear fluid, they were present to the number of 39 per cent. The average in general paralysis, in the living cases, was 9 per cent., and in post-mortem cases only 1 per cent. This is difficult to explain, but apparently they are more constant in general paralysis than in other conditions. We feel satisfied that the presence of polymorphonuclears in any large numbers can usually be accounted for by blood contamination, and can therefore attach no definite significance to their presence. In arteriosclerotic dementia they were present in nine fluids, varying from one to five to a count. One case of "cloudy" fluid showed 4 cells. The average for ten cases was 2.5. In one case, marked "bloody," they were present to the number of 64 per cent., and the count was 120 cells. In post-mortem fluids of arteriosclerotic they were absent in three fluids that were clear, and 16 per cent. were present in one count where the fluid was "bloody." They were absent in three cases of senile dementia, all "clear." In alcoholic (acute) condition they were absent in one case, "clear," present in two cases,—8 and 12 respectively,—the former "turbid" and the latter "clear." Their presence in such small numbers can have no special significance. It is possible with a poorly stained specimen to confuse these cells with endothelial cells, and that probably accounts for the fact that Cornell gave them such an importance in paralytic dementia. The question of mononuclears has been previously discussed, and we would emphasize the fact that where so few polymorphonuclears are found, still fewer mononuclears are to be found, and one must be suspicious of counting them as such; Cornell speaks constantly of mononuclear increase and is not clear in regard to the same.

7. UNDIFFERENTIATED CELLS.

In this class we have placed cells that we found, that did not conform to the types above described. In some instances they may be altered forms of the cells described, or degenerate forms, and in some cases we have found cells that resembled the fibroblasts (Plate 17, Fig. 14) found in the pia. These cells in the fluid are distinguished by their large oval or spindle-shaped nuclei with sparse and faintly-staining chromatic granules, but relatively slight amount of faintly pink-stained protoplasm, often only seen at the

poles of the cell. They were present in small numbers in almost all of our cases of general paralysis and organic dementia post-mortem, and in some cases of general paralysis ante-mortem. Their significance at present is not clear; they seem to be of no great importance in diagnosis. They seem to take part in the general cell increase in the fluid post-mortem. Cells possibly ependymal in origin are also here included.

DIFFERENTIAL COUNTS IN VARIOUS PSYCHOSES.

(Charts I. and II.)

Differential counts were made in all cases where the total cell count was over 50 to 100 fields. We will consider here the fluids of living cases only, as the value of the count for diagnostic purposes is the most important feature of the work. As would be expected from what we have said, the count is most important in general paralysis. Here we get positive findings; in all other conditions examined by us the cell findings can be considered negative. The great difference between the counts in general paralysis and other psychoses can be seen at a glance on Chart II. The total count in general paralysis varies from 110 to 1500 (the average being 450). The following proportions are taken from the average of nineteen cases:—

Lymphocytes, 73 per cent.
Endothelial cells, 13 per cent.
Plasma cells, 2 per cent.
Phagocytes (in four cases), 1 per cent.
Polymorphonuclears, 9 per cent.
Unclassified, 2 per cent.

So we see that the total number of cells is due in a large measure to a true lymphocytosis.

From our work so far, we can say that a lymphocytosis always occurs in general paralysis, and as a diagnostic factor is of the utmost importance. When the total count is over 100 per 100 fields, and contamination by blood eliminated, it is almost safe to say that the puncture is diagnostic of general paralysis. The presence of plasma cells even in so small amount as 1 per cent. is the strongest evidence of general paralysis, and confirms the evidence of the lymphocytosis, so that the lymphocytosis and presence of plasma cells together establish the diagnosis. In other conditions we found often a high

cell count, but the differential count showed that the large count was due to some other cause than a lymphocytosis.

In case No. 43 the count was 120, but only 31 per cent. lymphocytes, 5 per cent. endothelial, and 64 per cent. polymorphonuclears. Here a suspicious plasma cell was found, but the fluid was extremely bloody, so that we considered the puncture negative. The clinical diagnosis was organic dementia (hemiplegic). In another case (No. 34), with a clinical diagnosis of general paralysis (?), the total count was only 80, and differential count as follows: lymphocytes, 37 per cent. ; endothelial cells, 30 per cent. ; polymorphonuclear, 30 per cent. In this case the fluid was bloody. As these two cases have not come to autopsy, we cannot justify our diagnosis made upon the evidence of the findings in cerebro-spinal fluid, but from the findings in our other cases of general paralysis, four of which were confirmed at autopsy, we feel reasonably sure of calling the above two cases negative.

It is surprising to see the difference between the total cell count in other conditions and that in general paralysis. In no other series of cases did the count approach that in general paralysis, except where the fluid was bloody and the large cell count could be explained by an increase in polymorphonuclears. By consulting Chart II. the counts in the various conditions can be easily compared with those in general paralysis. In dementia præcox there is apparently an increase of lymphocytes, not to the same extent as in general paralysis, and we are unable to explain this fact. Especially is this true of the catatonic forms and in cases of many years duration. Thus in one case (No. 66), of fourteen years duration—a profoundly demented person—the total count was 150 cells for 100 fields: 74 per cent. lymphocytes, 22 per cent. endothelial, and 4 per cent. unclassified. In another case (No. 29), of six years duration—in a catatonic stupor—the cell count was 290: 84 per cent. lymphocytes, 12 per cent. endothelial, and 4 per cent. polymorphonuclears (clear fluid). The absence of plasma cells differentiates these fluids from that of general paralysis. In early cases of dementia præcox, however, where the lumbar puncture would be of importance as an aid to diagnosis, there seems to be no increase in lymphocytes.

CORRELATION WITH AUTOPSY FINDINGS.

Chart III. shows in a general way the proportion of cells found in the pia compared with the same elements as found in the cerebro-

1

CHART II.
Summary of Cell Counts. Ante-mortem Fluids.

No. of Fluids.	Diagnosis.	Cells in 100 Fields. Average.	Lymphocytes. Per cent.	Endothelial Cells. Per cent.	Plasma Cells. Per cent.	Phagocytes. Per cent.	Polymorpho-nuclear leucocytes. Per cent.	Other Cells. + Unclassified. Per cent.
19	General paralysis.	110-1500	73	13	2	1% of 4 cases	9	3% of 7 cases
10	Organic dementia	8-80	+	+	+	...
3	Senile dementia	7-81	+	+	+	...
2	Polynuclear delirium	20-24	+	+	+	...
1	Chr. alcoholic insanity	...	+	+	+	+
4	Epilepsy	12-52	+	+	+	...
4	Manic-depressive.	11-49	+	+
1	Involution melancholia	...	57	40	86	3
9	Dementia præcox	14-290	74	26	3	...
1	Cerebral lues	...	+	+	...
1	Neurasthenia	...	31	5	1?	...	64 (bloody)	...
1	Organic dementia?	...	37	30	30 (bloody)	...
1	General paresis?

+ Indicates that cells were present in these conditions, but in such a small number that percentages were valueless.

Summary of Cell Counts. Post-mortem Fluids.

No. of Fluids.	Diagnosis.	Cells in 100 Fields. Average.	Lymphocytes. Per cent.	Endothelial Cells. Per cent.	Plasma Cells. Per cent.	Phagocytes. Per cent.	Polymorpho-leucocytes. Per cent.	Other Cells. + Unclassified. Per cent.	Körnchen Cells. Per cent.
12	General paralysis	500-3400	62	24	5	3 (9 cases)	1 (7 cases)	5 (6 cases)	
8	Organic dementia	320-2157	19	68	(2 cells? in 2 cases)	5 cells in 3 cases	1 (4 cases)	6 (7 cases)	(10% in 1 case)
3	Senile dementia	340-800	26	63	(2 cells? in 2 cases)	10	
1	Manic-depressive	960	23	75	...	1	...	1	
1	Chr. alcoholic insanity	86	60	32	3	5	
1	Dementia præcox	8	+	+	
1	Paranoia	156	54	41	5	
1	Paralysis agitans	800	10	49	32	9	
2	Idiotic paraplegia	64-520	6	90	4	...	
1	Morphinism	100	44	46	10	...	
1	Toxic delirium	54	29	39	+	32	1 in 1 case
1	Organic dementia?	45	+	+	+	+	
1	General paresis?	556	66	30	...	4	

N.B.—Note the uniform increase in the total number of cells in the post-mortem fluids in the majority of psychoses.

CHART III.

Correlation of Cells in Fluids and Pia.

Pathological Findings.	(1) Fluid Examined. (2) Pia Examined.	Plasma Cells.	Endothelial Cells.	Phagocytes.	Fibroblasts.	Lymphocytes.	Poly-morpho-nuclear Cells.	Mast Cells.	Un-classified.
General paresis.	Fluid, No. 1 Autopsy, No. 1188. Cerebellum Lt. precentral . . .	8% Many Many (frequently with double nuclei) Many ...	19% Few Numerous	6% One Two	... Few Many	66% Many Many	1%
	Dorsal cord Choroidal ependyma	" None	Many ...	Many
	Fluid, No. 2 Autopsy, No. 1189. Rt. sup. frontal	8% Many (some multi- nuclear) Many	19% Many	6% None	... Some	59% ...	1%
General paresis.	Rt. precentral	Numerous (small variety)	"	"	Many
	Dorsal cord Lumbar cord . . .	" "	" "	" "	" "	" "
	Fluid, No. 7 Autopsy, No. 1190. Cortex .	10% Many	24% Numerous	1% None	65% Many
General paresis.	Fluid, No. 51 . . .	9% 6% Many	44% 8% Many	1% ... One	... Numerous	48% 78% Many	... 4%	8% 8% ...
	" No. 8 Autopsy, No. 1184. Lt. sup. front. Rt. sup. frontal . . .	" "	" "	0 0	" "	" "
	Cerebellum Floor of IV. ventricle Dorsal cord . . .	" ... Many	" ... Very few	0 0 0	" "	" "
General paresis.	Fluid, No. 28 . . .	3% Many Numerous	40% Many Few	10% 0 0	46% Many ...	1%
	Autopsy, No. 1192. Cortex Cervical cord
	Fluid, No. 41 . . .	7% 2% Many	3% 4% Numerous	... One	78% 94% Many	1% Two	11%
General paresis.	" No. 26 Autopsy, No. 1193. Cerebellum Lt. frontal . . .	" "	" "	0 0	... Numerous	" "
	Rt. precentral . . .	" "	" "	0 0	...	" "
	Cord . . .	" "	" "	0 0	Numerous	" "

Cerebral arterio-sclerosis Chronic lepto-meningitis	Fluid, No. 80 Autopsy, No. 1197.	Cortex :	1% (?) 0	50% Many	Several	Present	39% Few	1%	9% ...
General paresis .	Fluid, No. 25 Autopsy, No. 1206.	Rt. frontal	2% Many (some with double nuclei)	30% Many + (frequently pig- ment laden)	2% 0	...	64% Many	2% ...
Cortical atrophy ?	Fluid, No. 17 No. 94 Autopsy, No. 1202. Rt. frontal 0	67%	+ 14% Few	19% ...
Thrombosis, as- cending parie- tal artery with softening	Fluid, No. 93 Autopsy, No. 1201.	. . . Rt. pre- central	... One (?)	75% Many	1%	23% 0	1% ...
Cortical soften- ings	Fluid, No. 182 Autopsy, No. 1199	84%	9% ...	7%
General paresis .	Fluid, No. 96 Autopsy, No. 1203.	Rt. frontal	3% Many	22% ...	1% 0	...	62% Few	2%	10% ...
.	Rt. precentral Cord Present	" "	...	0 0	...	" "
Normal brain and cord	Fluid, No. 103 Autopsy, No. 1204.	. . . Cord	...	84% ...	1% (?)	7% ...	1%	7% ...
Normal brain and cord	Lt. precentral Rt. frontal	Increased "	0	SI. increase "
Cerebral arterio- sclerosis	Fluid, No. 91 Autopsy, No. 1200.	. . . Rt. frontal	... 0	46% Few	... 0	Increased	44% Rare	10%
	Fluid, No. 82 Autopsy, No. 1198.	. . . Lt. pre- central	... 0	49% Few	... 0	Increased	10% Few	32%	9% ...

Part of pia examined
normal.
Areas of softening
not examined.

Normal pia.

Strictly speaking the pathological lesions found in cases marked "general paresis" were those of chronic exudative meningo-encephalitis, but when taken together with the clinical picture the former diagnosis seems justified.

spinal fluid. By glancing over this table it is evident that the pia mater is the origin of most of the cells found in the fluid, and that the numbers are in a general way correlated.

Here, again, the plasma cell is worthy of the greatest consideration. Numerous plasma cells were found in the pia of every section examined in cases of general paralysis, whether cortex or cord, and, as a rule, sections of the pia as far apart as possible were taken for this purpose. In our series only in otherwise clearly defined cases of general paralysis were plasma cells definitely found, and in all of these cases, whether living or dead, the cerebro-spinal fluid contained plasma cells.

A very few phagocytic endothelial cells were found in the pia of those cases of general paralysis of the eight autopsies, whereas they appeared in the fluid of seven cases. Doubtless further search would have shown a greater number in the pia. As phagocytes also occurred in the post-mortem tissues, we cannot attach as great significance to them as to the plasma cells.

Polymorphonuclear leucocytes were not demonstrated in the pia of our cases, though it is well known that they do occur in rapidly progressing cases.

Pial lymphocytosis in these cases is quite characteristic of general paralysis, and corresponds very closely with the degree of lymphocytosis in the fluid. Endothelial cell proliferation seems to be a common occurrence in a variety of conditions.

So we find that in the pia of the cases which came to autopsy are found approximately the same kind and number of cells which appear in the corresponding fluids, and notably that in general paralysis the pia, showing an excess of lymphocytes, is without doubt the seat of origin of the same cells in the cerebro-spinal fluid.

In conclusion we desire to extend our sincere thanks to Dr Chas. W. Page, Superintendent of the Danvers Insane Hospital, for his kindly interest and encouragement, and to Dr F. B. Mallory and Dr Adolf Meyer for their valuable assistance in the identification of the various cell types, to Dr F. P. Gay for many valuable suggestions, and to our colleagues on the Staff of the Danvers Insane Hospital, and the assistants in the laboratory, who were always ready and willing to co-operate with us in this investigation.

CYTOLOGICAL STUDY OF CEREBRO-SPINAL FLUID 227

SUMMARY.

1. We cannot but regard Alzheimer's method as the best yet devised for the cytological study of the cerebro-spinal fluid, the good results depending upon rapid fixation of the cells and the subsequent treatment of them as if they were tissue.

2. A good differential count and a fair quantitative count are possible by this method.

3. The cells regarded by us as of greatest diagnostic importance are the plasma cell, the phagocytic endothelial cell, the fatty granule cell, and the lymphocyte if in excess.

4. In psychiatry the cell picture in general paralysis stands out distinctly from that in the other forms of insanity, the latter being considered by us as presenting nearly normal fluids.

A high cell count, with excess of lymphocytes, over 100 to 100 fields, the presence of plasma cells and perhaps phagocytes, in a case of suspected general paralysis, is the strongest evidence in favour of this diagnosis.

5. It is possible that other organic cerebral conditions may show a cell picture of diagnostic importance, as indicated by the finding of fatty-granule cells in these conditions post-mortem.

6. The origin of the cells in the fluid is without doubt in large measure, if not entirely, traced to the pia mater.

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DESCRIPTION OF PLATES.

PLATES 15 and 16.—Photomicrographs of cells found in cerebro-spinal fluid.
Pyronin stain : $\times 1500$ diameters.

Fig. 1 A.—Lymphocyte. Note the absence of protoplasm and distinct chromatophilic granules, "clock-face" arrangement.

228 CYTOLOGICAL STUDY OF CEREBRO-SPINAL FLUID

B 1, 2, 3.—Various types of endothelial cells.

B 1.—Large endothelial cell, the nucleus swollen, and taking up most of the cell. Very little protoplasm visible. The nucleus is folded upon itself, but the thin line between the halves does not show, and the nucleus has a round appearance.

B 2.—The usual type of endothelial cells. Horse-shoe shaped nucleus beset around the periphery of the cell, with very little protoplasm visible.

B 3.—Small type of endothelial cells, sharply indented nucleus. Very few chromatophilic granules. Note the absence of distinct granules in all nuclei of endothelial cells as compared with the nucleus of phagocytes.

Fig. 2.—Phagocytizing endothelial cell with lymphocyte inclusion. The nucleus is flattened and crowded to the periphery. Note the light court around the lymphocyte and enormous swelling of the protoplasm of the endothelial cells.

Fig. 3.—Plasma cell. Rather indistinct, but dark staining nucleus with "clock-face" arrangement of granules can be made out, also the shape of protoplasm, with a brighter area in the lower part. Protoplasm a deep red and nucleus a deep blue. (See Plate II., 6, 7, 8, 9.)

Fig. 4.—Cell showing mitotic figure in active state of karyokinesis. Difficult to determine to which type this cell belongs.

Fig. 5.—"Körnchen cell." The nucleus is hardly visible, but the fat droplets can be distinctly seen.

Fig. 6.—Low power field in case of general paralysis, showing the distribution and density of cells. The total cell count here was 900 to 100 fields. A normal field would show only one or two cells at the most.

PLATE 17.—Cells of cerebro-spinal fluid. Pyronin stain : drawn with camera lucida (No. 4 ocular and $\frac{1}{2}$ oil immersion).

Figs. 1, 2, 3.—Phagocytes. In 1 and 3 the inclusions are lymphocytes, but in fig. 2 the inclusion is apparently an endothelial cell, or perhaps a degenerated lymphocyte.

Figs. 4, 5.—Endothelial cells.

Figs. 6, 7, 8, 9.—Plasma cells.

Fig. 10.—Transitional form (?) between lymphocyte and plasma cell.

Fig. 11.—Lymphocyte.

Fig. 12.—Mitotic figure in cell, whether plasma or endothelial not decided.

Fig. 13.—"Körnchen" cell.

Fig. 14.—Fibroblast (?).

Fig. 15.—Polymorphonuclear lymphocyte. No protoplasm visible.



FIG. 1.



FIG. 2.

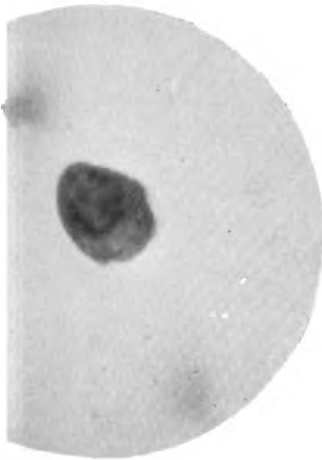


FIG. 3.



FIG. 4.

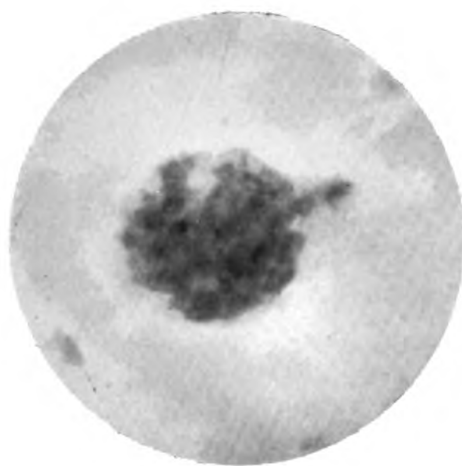


FIG. 5.

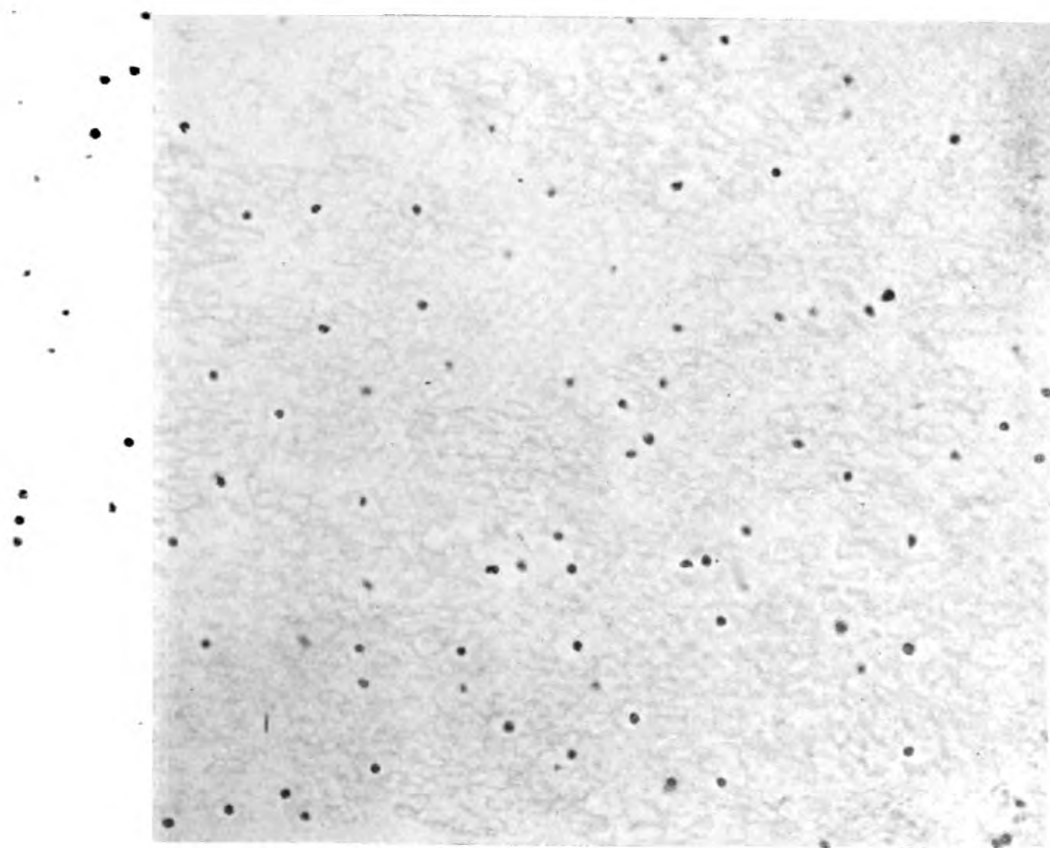
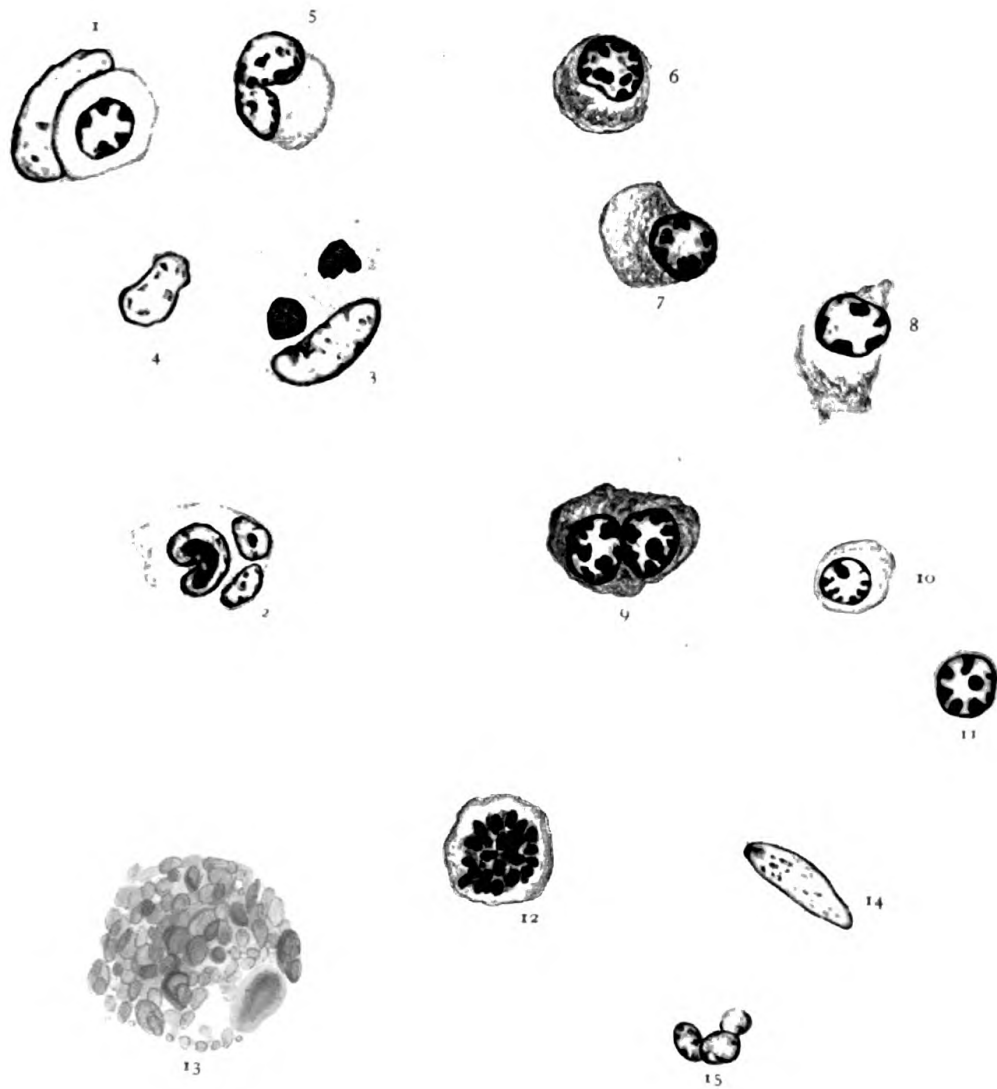


FIG. 6.



- | | |
|----------------------------|------------------------------------|
| 1, 2, 3. Phagocytes. | 12. Mitotic Figure (Plasma Cell?). |
| 4, 5. Endothelial Cells. | 13. Körnchen Cell. |
| 6, 7, 8, 9. Plasma Cells. | 14. Fibroblast (?). |
| 10. Transitional Cell (?). | 15. Polymorphonuclear Leucocyte. |
| 11. Lymphocyte. | |

All cells stained with pyronin-methyl green.

EXOPHTHALMIC GOITRE COMBINED WITH MYASTHENIA GRAVIS.

By GEORGE E. RENNIE, M.D., F.R.C.P. Lond.,
Physician to the Royal Prince Alfred Hospital, Sydney.

THE combination of the symptoms of Graves' disease with those of myasthenia gravis does not appear from the literature to be a common one, in spite of the assertion of Meyerstein (1) to the contrary. I have only been able to find recorded, in addition to Meyerstein's case, two cases by Loeser (2) and one by Brissaud and Bauer (3). The association of the symptoms characteristic of these two diseases in one individual patient is sufficiently striking to be recorded. The following case of this nature was admitted under my care on September 11, 1907, in the Royal Prince Alfred Hospital, Sydney.

The patient was a single man, 27 years of age, a blacksmith by occupation, who had always lived in a country town in New South Wales, and whose life, according to all accounts, had been an exemplary one. He had been a good, hard-working son, and had always been temperate so far as alcohol was concerned, a moderate smoker, and had never suffered from any venereal infection.

His family history showed nothing remarkable. His father died from a paralytic shock; his mother is healthy, except for some slight rheumatism. Nothing could be ascertained as to the existence of any disease in near relatives which could throw any light on his condition.

He had always lived in the same town in which he had been born, in a healthy part of the country, which is dry and hot.

So far as could be ascertained, his illness began about three years ago with symptoms suggestive of diabetes. He first noticed that he was eating more than usual, and also taking a lot of fluid, as his mouth and tongue were very dry, and he began to get very weak. He then left his occupation—that of a blacksmith—because he did not feel equal to the work. He took to droving, and for the last three years has been subject to

much exposure. Eighteen months ago he first noticed swelling in the neck, and twelve months ago noticed that his hands trembled. Eight months ago he noticed that his eyes were becoming more prominent, and soon after that his right eyelid began to droop, and shortly afterwards the same condition was noticed in the left eyelid. Six months ago he noticed weakness in his right arm; then in his right leg; then in the left leg and left arm in that order. He noticed that he got tired very easily, but after a short rest was able to go about his work again.

On examination his intelligence is normal. He has no headache and no pain of any kind. He has no fits or faints, and his speech is normal. It is noticed that his speech does not become nasal in character, nor does he become tired after talking. His hearing and smell are normal; he has occasional diplopia; there is marked exophthalmos of both eyes, and bilateral ptosis which is but slightly marked in the morning, but which becomes more marked towards night. The pupils are moderately dilated, but react to light and on convergence.

There is marked external ophthalmoplegia in both eyes, the movements of the eyeballs being very limited in all directions, but the amount of movement varies to a slight extent from day to day. The face is smooth and somewhat expressionless. All the facial movements are limited, but are equal on both sides. The angles of the mouth tend to droop, and the "nasal smile" is characteristic. There is no difficulty in swallowing; no regurgitation of fluid through the nose; no paralysis of the palate; the tongue is protruded in the middle line; the muscles of the jaw are weak, and become rapidly exhausted during the mastication of food.

There is marked weakness in all the muscles of the trunk, but there is no difficulty in maintaining the head erect. The muscles of the limbs are all weak; the extensors of the forearms and arms are more affected than the flexors; and the muscles of the right arm and leg are somewhat weaker than those of the left. All these muscles become easily exhausted on exertion. For instance, when the patient is taking his food he can raise his right arm to his mouth two or three times, and then is exhausted and has to use his left arm to assist the right.

The wrist and elbow jerks are difficult to elicit. The knee-jerks are present on both sides, though somewhat diminished, and

become exhausted after twenty or thirty taps. But after a rest of a few seconds they become more active than before. The muscles of the face, arm, and leg, on being stimulated by a faradic current, become exhausted in about twelve to fifteen seconds, but recover their excitability after half a minute and become as active as before. The sphincters are normal, and no alteration in sensation could be elicited.

The thyroid gland is moderately enlarged, but no enlargement of the thymus could be made out on percussion. The heart sounds were normal, but his pulse rate varied from 96 to 124, most frequently being 108. His temperature during the time of his stay in hospital was irregular; the first few weeks it ranged from 97° to 100°, but during the latter part of his stay kept nearer the normal line. He had no frequency of micturition and no pain in passing water. The quantity of urine averaged about two pints in twenty-four hours. The urine was always acid, the specific gravity varying from 1020 to 1026, contained no albumen, but at times gave the reaction for sugar. His appetite was fair, he was not thirsty, his tongue generally fairly clean, and his bowels acted regularly every day without aperient. His body weight varied very slightly, but on leaving the hospital he was 1½ lbs. heavier than on admission. His blood examination showed: red cells, 4,100,000; white cells, 8100; Hb. value, 80 per cent.; polymorphs, 34 per cent.; large lymphocytes, 48 per cent.; small lymphocytes, 12.9 per cent.; eosinophiles, 3 per cent.; mononuclears, 5 per cent.; mast cells, 1 per cent.

During his stay in hospital he was treated with strychnine and tincture of belladonna, the latter being given in gradually increased doses up to 27 minims three times daily; but with the exception of the thyroid gland being slightly reduced in size, and the exophthalmos being slightly less marked, no appreciable effect in his condition was produced.

I think it must be admitted that among the symptoms above described we have those typical of Graves' disease, viz., exophthalmos and enlarged thyroid gland and tachycardia. Then the symptoms of myasthenia gravis; the ocular paralysis, the ptosis varying in amount—worse towards night and better in the early morning—the smoothness of the face, and the "nasal smile," the rapid exhaustion of the muscles of the jaws and tongue on eating, the exhaustion of the muscles of the arms

and legs, the exhaustion of the deep reflexes, and the faradic myasthenic reaction, are all characteristic of this disease. The intermittent glycosuria is a symptom which may be associated with either disease.

Meyerstein's case showed, in addition to the typical symptoms of myasthenia, such of Graves' disease as : exophthalmos, enlarged thyroid, tachycardia, vaso-motor disturbances, etc. He remarks that " the symptoms of Graves' disease appear to occur not rarely in myasthenia gravis, so that the concurrence does not appear to be accidental—that is, both symptom complexes may follow from the same noxa ; or Graves' disease may favour the development of myasthenia."

Loeser discusses two cases of myasthenia which were combined with Graves' disease. In both cases, during the course of a year, typical myasthenia had developed on the basis of Graves' disease.

The questions that present themselves in considering this case are:—Is there in this patient the operation of two etiological factors? Are the symptoms of Graves' disease due to perverted thyroid function, and the myasthenic symptoms to some hypothetical toxin? Or, is there but one morbid factor at work which is producing all the symptoms enumerated? In other words, could his myasthenic symptoms be produced by the perverted thyroid secretion? Or, could his Graves' symptoms be interpreted in any way as the result of the operation of the toxin of myasthenia gravis?

In view of the fact that Graves' disease is comparatively common, and the association of the symptoms of myasthenia gravis with it is rare, it seems difficult to believe that the latter can be caused by the perverted thyroid secretion, more especially since Buzzard (4), in the cases of myasthenia gravis described by him, failed to find any evidence of an associated lesion of the thyroid gland. In this connection it is interesting to note that the first case of myasthenia gravis I saw, and the first case I believe to have been reported in Australasia (5), occurred in a jockey, whose symptoms developed somewhat rapidly after taking very large doses of thyroid gland tablets for the purpose of reducing his weight.

In the meantime, however, we cannot arrive at any definite solution of these questions.

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Abstracts

ANATOMY.

STUDIES IN NERVE CELLS. (1. The Molluscan Nerve Cell, together (181) with Summaries of recent Literature on the Cytology of Invertebrate Nerve Cells.) W. M. SMALLWOOD and C. G. ROGERS, *Journ. Comp. Neur. and Psychol.*, Jan. 1908, p. 45.

THIS is an elaborate paper dealing with the structure of invertebrate nerve cells, more especially in the gastropoda, but exclusive of the neuro-fibrillæ. It is well illustrated, and there is a large bibliography. The author's own summary is quoted.

(1) The nervous system of gasteropods does not permit of direct stimulation of a specific ganglion because of the compactness of the nerve collar and the numerous nerves arising from the different ganglia.

(2) Lymph canals are not identical with the cytoplasmic vacuoles. They really exist, and have a rather general distribution among the nerve cells of invertebrates.

(3) Vacuoles are present in the cytoplasm of nerve cells of Nemerteans, Annelida, Crustacea, Insecta and Mollusca. The vacuoles can easily be seen in the living cells as independent structure filled with a fluid or differential bodies. They are transitory structures, vary in number, and are not limited by distinct walls.

(4) Nissl bodies exist in invertebrate as well as vertebrate nerve cells. They are found to occupy a zone of cytoplasm next to the nucleus, but not extending out to the cell wall in most instances. They are chiefly arranged in rows or in spindle-shaped groups. The Nissl bodies are aggregates of extremely minute particles and exhibit marked resistance to degenerative changes. They actually

exist in the living nerve cell. Those occurring in *Limax* are always found within the limits of the cytoplasmic vacuoles. They can be caused to appear in the cell by rest and feeding, and can be made to disappear through hibernation, fatigue and electrical stimulation. They are probably of a fatty nature.

(5) Pigment granules are found very generally in molluscan nerve cells. They do not readily respond to starvation experiments, can be increased in size and number through feeding, are practically unchanged by fatigue on electrical stimulation, but do show occasional variations in size and number. These bodies respond to the tests for lipochrome substances or fats.

(6) The centrosome has been described in many of the invertebrate nerve cells, but there is considerable doubt as to its persistent presence in adult nerve cells.

J. H. HARVEY PIRIE.

REMARKS ON THE PRESENT POSITION OF THE NEURONE

(182) **THEORY.** (*Bemerkungen zum heutigen Stand der Neuronlehre.*) MAX VERWORN, *Med. Klinik*, No. 4, 1908, p. 111.

THIS article is a concise review of the present position of the neurone theory. It contains no original matter. In support of the neurone theory, to which the author adheres, he quotes the work of Cajal, Harrison, and others. He indicates the need for further observations with regard to (1) the mode of connection of neurones with each other, (2) the relative amount of nutritional influence exerted on a peripheral nerve on the one hand by its ganglion cell and on the other by its immediate surroundings, (3) the parts played in nervous conduction by the neurofibrils and the perifibrillary protoplasm, and (4) the process of inhibition. He discusses briefly some of the more important views held as regards these subjects.

ALEXANDER BRUCE.

HYPOSPINAL MICRO-SYMPATHETIC GANGLIA. (*Über die mikro-*

(183) *sympathischer, hypospinalen Ganglion.*) MARINESCO and MINEA, *Neurolog. Centralbl.*, Feb. 15, 1908, p. 146.

WHILE studying the posterior root ganglia in serial sections stained by Cajal's method, the authors have discovered what they believe to be hitherto undescribed ganglia. These lie in the fat outside or below the posterior root ganglia; they have never found them on the proximal side. Usually microscopic, they may sometimes be large enough to be seen by the naked eye in sections. They

may be single or multiple, as many as eight having been noted in relation to one nerve root; their size is inversely proportional to their number. A ramus communicans has pretty often been traced to the infra-ganglionic part of the nerve root. This may be so short that these little ganglia lie in direct contact with the lower pole of the root ganglion, or it may be of some length, in which case they are to be looked for in close relationship to the ramus communicans from the sympathetic cord. Microscopically they contain all the three cell types found in the large sympathetic ganglia: (1) cells with short intra-capsular dendrites and one axis-cylinder process; (2) large cells with long forking extra-capsular dendrites and one axis-cylinder process. These are not abundant, however; (3) cells of a type intermediate between these two.

The authors regard these hypospinal ganglia as arising in the same manner as the ganglia of the sympathetic cord, the ventral separation from the root ganglia simply not occurring to the same extent. As regards the destination of the fibres arising from the cells of these hypospinal ganglia, they offer no opinion as to whether they join the pre-vertebral sympathetic cord, or serve surrounding tissues, or have some other function.

J. H. HARVEY PIRIE.

THE FLOOR OF THE FOURTH VENTRICLE. (*Le plancher du (184) quatrième ventricule. Étude morphologique.*) MINEFF MICHAEL, *Névrose*, Vol. ix., f. 2, 1908, p. 115.

THE author has studied, and illustrates with stereoscopic photographs, the surface form of the floor of the 4th ventricle in forty-nine human brains, of which forty were adult and nine foetal.

The average width in adults is 16 mms., and the length 34 mms.

Analysing the different formations, he finds:—

(1) The medullary striæ conformed to the description given by Retzius.

(2) Trigonum hypoglossi—to consist of three parts:

(a) a mesial area;

(b) an area plumiformis, showing oblique striæ;

(c) an outer triangular area, to which the name of trigonum hypoglossi should be reserved.

(3) Trigonum vagi varies in form, and the areas of opposite sides may be connected together by an intercinereal commissure.

(4) Funiculus separans is merely a local thickening of ependymal neuroglia, but is useful for description.

(5) Area postrema is an area situated on either side of the

mesial plane at the posterior end of the calamus, with a grey brown surface slightly mammillated. The inner margins pass down to the entrance to the central canal, and the pia is adherent along the outer margin. The true obex is seldom present, but constitutes a bridge uniting the two areas of opposite sides.

(6) Trigonum acousticum.

(7) Upper part of the floor.

(8) Median furrow.

These parts are carefully described, and compared with the views of other writers, and it is shown that many variations are found.

DAVID WATERSTON.

THE CONDITION OF THE NORMAL COCHLEAR NERVE IN

(185) **THE INTERNAL AUDITORY MEATUS.** (*Das Verhalten des normalen Nervus Cochlearis im Meatus Auditorius Internus.*) S. ALEXANDER and D. H. OBSTEINER, *Zeitschr. f. Ohrenheilk.*, Bd. 55, Heft 1-2, 1908, S. 78.

TEN years ago Obsteiner reported on a specimen sent to him by Siebenmann in which small round bodies, staining blue with hæmatoxylin, were observed in the sections of the auditory nerve. On longitudinal or oblique sections these bodies were arranged round the central part of the nerve which takes on the eosin stain more faintly than the peripheral part and appears as a cone-shaped structure—the apex of the cone being towards the internal ear. At the time Obsteiner was in doubt as to the nature of these bodies, but thought that they were either pathological or post-mortem artefacts. Lately Alexander and Obsteiner have examined ninety-two specimens of the auditory nerve from about fifty patients, whose ages varied from an eight months' foetus up to ninety-four years. In the younger subjects these bodies, which both observers regard as corpora amylacea, were never found. They were first found in the auditory nerves of patients aged twenty-six, and thereafter the older the subjects were the more frequently the bodies were observed. Staining reactions proved them to be corpora amylacea. Similar appearances have been found by Obsteiner in the posterior spinal nerve roots. The faintly stained region lies between the part of the nerve which possesses the sheath of Schwann and the part surrounded by neuroglia—in other words, where peripheral changes to central. The motor roots of the spinal nerves and also the motor nerves springing from the medulla show entirely the peripheral formation. The work of Nager in Siebenmann's Klinik and of Hülles is referred to. Alexander and Obsteiner conclude that the blue staining bodies are corpora amylacea occurring at the junction of the two

parts (peripheral and central) of the auditory nerve, and emphasize the importance of cutting this nerve as near as possible to the medulla when the temporal bone is removed for microscopic investigation, and of obtaining the specimens as soon as possible after death.

J. S. FRASER.

PHYSIOLOGY.

STUDIES IN REFLEXES: THE BEHAVIOUR OF SOME (186) CUTANEOUS AND TENDON REFLEXES IN INFANCY.

(Reflexstudien: Ueber das Verhalten einiger Haut- und Sehnenreflexe bei Kindern im Laufe der ersten Lebensjahre.)

Z. BYCHOWSKI, *Deutsche Ztschr. f. Nervenheilk.*, 1908, Bd. xxxiv., Heft 2, S. 116.

BYCHOWSKI finds that the various cutaneous and deep reflexes do not all develop simultaneously, and that they are of varying phylogenetic importance. Thus if reflexes A, B and C develop at different periods, and are probably separated from each other by considerable intervals, we should expect that in a given individual their times of appearance should also differ. He has studied the question as to how during the first few months of life those reflexes behave which in the adult are constantly present; or, in other words, does an infant come into the world with all its reflexes in full activity or not? Bychowski has examined about a hundred infants, noting the condition of the knee-jerks and ankle-jerks, and of the abdominal and cremasteric reflexes. He has also studied the plantar reflex; but owing to the complex motor phenomena produced, he has excluded this reflex from his paper. He selected healthy infants, testing their various reflexes during the waking condition and in a good light. Crying children were not examined, since in such a state the abdominal reflexes are difficult to elicit. He gives a tabular account of the reflexes in sixty-seven healthy infants under one year old. He also examined a number of children during the second year of life. The knee-jerk was constantly present in all of these cases, with only two exceptions, in both of which the children were ill. The knee-jerk seemed, as a rule, to be livelier than in the adult. Sometimes a consensual knee-jerk of the opposite limb was also present. The ankle-jerk, on the contrary, was absent in 60 out of 64 patients of six months and under: in the remaining case, from nine to ten months of age, it was present twice out of thrice. The ankle-jerk is first elicited about the fifth or sixth month, and becomes constantly present at the beginning of the second year. In one child the ankle-jerk was tested every two days from the

time of birth, until at last, at the age of seven months, the ankle-jerk appeared. Fuhrmann, on the other hand, found the ankle-jerk absent only in 34 per cent. of children examined during the first week of life. Whether Bychowski or Fuhrmann be the more accurate observer, the fact remains that the ankle-jerk is less constantly present than is the knee-jerk.

Next, as to the abdominal reflexes, which in healthy adults are constantly present (excluding, of course, multiparous women and other cases with pendulous bellies); the cutaneous reflexes, it should be borne in mind, are more easily exhausted than are the deep reflexes. Bychowski found all four abdominal reflexes (upper and lower on each side) present in sixteen infants out of sixty-seven; in seven cases the upper and the lower were present alone. The abdominal reflexes are not present at birth. Fuhrmann's statistics also support this view, inasmuch as he found absence of the abdominal reflexes in 80 per cent. of infants during the first month, in 65 per cent. during the second month, in 45 per cent. during the third month, and in 50 per cent. during the fourth month. Whichever statistics be accurate, it is evident from both observers that the abdominal reflexes are of lower phylogenetic importance than the deep reflexes.

As to the cremasteric reflex, it was absent in eleven cases out of twenty-six, being practically constant after the age of four months.

Summing up, then, the knee-jerk is the only one of the above reflexes which is constantly present from birth. It is therefore of greater phylogenetic importance than the other.

PURVES STEWART.

DOES THE HUMAN RECURRENT LARYNGEAL NERVE CON-
(187) TAIN SENSORY FIBRES? (Führt der Rekurrens des
Menschen sensible Fasern?) A. KUTTNER and E. MEYER
 (Berlin), *Archiv für Laryngologie*, Bd. 20, Heft 2, S. 356.

THIS short paper is only the latest stage of a dispute that has been running in these Archives for more than a year on the so-called "Recurrent Question." Grossmann, Katzenstein, Réthi, Schulz, and others proved that in cats, rabbits, goats and monkeys the recurrent laryngeal is a mixed nerve, whereas in the dog it is a purely motor nerve and only possesses a few sensory fibres by anastomosis. It was, however, impossible from the above experiments to draw conclusions as to its nature in man. Massei, in the Italian Archives of Laryngology, stated that peripheral recurrent paralyses were always connected with disturbance of sensibility of the larynx, but says that he never drew from this fact the conclusion that the recurrent was a mixed nerve. He

sought the explanation of the diminished sensibility rather in a communicating neuritis by way of the anastomosis existing between the superior and inferior laryngeal nerves. The sensibility of the larynx was investigated by means of the laryngeal sound, and, in cases of recurrent paralysis, Massei found that there was no reaction, or at most slight coughing and spasm, in marked contrast to the phenomena observed when normal individuals were investigated.

Kuttner and Meyer injected adrenalin and novocain into the region of the superior laryngeal nerve, and produced anæsthesia of the larynx, but Massei states that this disproves in no way the supposition that the inferior laryngeal is a mixed nerve; just as the proof that the inferior laryngeal is a mixed nerve would not upset the accepted rule that the superior laryngeal is the sensory nerve of the larynx.

Kuttner and Meyer have also examined numerous cases of recurrent paralysis by the method employed by Massei and have never been able to make out any anæsthesia: the patient not only felt each touch of the sound, but they reacted to it by reflex closure of the glottis and by coughing fits. The intensity of these reactions varied in these cases within the same limits as in healthy persons. Finally Kuttner and Meyer state that after section of the superior laryngeal nerve, even when the recurrent remains intact, reflex excitability is obliterated on the side of the larynx in question. During the course of this dispute the motor fibres of the recurrent have not escaped, as Kuttner and Grabower have written somewhat heated articles on Semon's law, Saundby's case and Grossman's explanation of the appearances in recurrent laryngeal paralysis. Altogether the "Recurrent Question" is still with us.

J. S. FRASER.

SENSITIVENESS TO LIGHT AND THE SIZE OF THE PUPIL.

(188) (*Lichtempfindlichkeit und Pupillenweite.*) H. S. LANGFELD,
Ztschr. f. Psychol. u. Physiol., Bd. 42, H. 5, 1908.

It is known that the size of the pupil is, in many persons, considerably above the average; this is especially noticeable by lamplight, and is more common in the female than the male sex. The author, having observed that these unusually large pupils also react sluggishly to light, was prompted to inquire whether such deviation in size was connected with any demonstrable peculiarity in the function of retina or iris.

The examinations were conducted on rather less than a score of persons.

The following possible causes of the abnormal size had first to be considered:—

(1) One of the subjects being blind to red rays, and the lamp with which the examinations were done being relatively rich in these rays, a connection between size of pupil and predominance of red rays might exist. But pupils equally large were present in those with normal colour-sense.

(2) Some subnormal sensitiveness or else unusual tendency to exhaustion, either in the light-perceiving organs, the centripetal pupillo-motor fibres, in the centres themselves, or in the reacting apparatus (musculature of iris)?

(3) Myopia?

A series of experiments were made on each of the subjects, and the results are tabulated.

The author tested the width of the pupil both by artificial light and in the dark, using in the first case an ordinary 10-candle-power incandescent light, and in the second case taking instantaneous photographs of the pupils by a flash-light apparatus, the illumination being sufficiently transient to fall entirely within the latent period of pupillary reaction.

The *mode of reaction* of the various pupils was then tested, a light-stimulus of easily variable intensity being employed. The author found it almost impossible to give the extent of reaction in figures, and contented himself with observing whether the degree of movement lay approximately within the normal limits. This occurred in all but one case. By use of the loup (corneal microscope) the great differences in energy and rapidity of reaction were easily observed.

Prof. W. A. Nagel's adaptometer was then employed in estimating the *light-sense* after adaptation (*a*) to ordinary daylight, (*b*) to darkness. In a certain number of cases the complicating factor produced by variations in the size of the pupil was excluded by employment of an "artificial pupil," which admitted the same amount of light into every eye.

The result of this series of investigations was to show, in the first place, that the unusually large size of the pupil which occurred in several of the persons observed had no connection either with the colour of the iris, with the refraction, with the colour-sense, or with the degree of sensitiveness to bright light (*Blendbarkeit*.)

The most striking difference in the size of the pupils is seen by lamplight—pupils which, in ordinary daylight, are of the same size, show by lamplight as much as $3\frac{1}{4}$ mm. of difference.

The pupils in one case showed a marked tendency to exhaustion, but the supposition that this might determine the large size of the pupil proved erroneous, as several equally large pupils exhibited a quick and lasting reaction.

Finally, as regards the *light-sense*, tested by the Adaptometer. The fact that the greatest differences in the size of the pupils were seen in faint lights, especially lamplight, suggested a deficient sensitiveness to faint light, or a diminished power of adaptation, in the persons with large pupils. The testing of this problem proved difficult.

The light-sense in every case was estimated (*a*) within the first minutes of exposure to darkness, (*b*) after twenty minutes in darkness. The figures yield nothing characteristic, and are really not comparable, since the amount of light entering each eye differed according to the size of the pupil, some pupils enlarging more than others on exposure to dark. As paralysis of the iris by homatropine or cocaine did not entirely equalise the size of the pupils, an artificial pupil of 2 mm. diameter had to be used. Even here the sensitiveness to light varied very greatly in the different cases, and gave little material for generalisation. The author, however, is inclined to conclude that in these persons who have narrow pupils (other things, such as age and refraction, being equal), adaptation to darkness occurs somewhat more promptly and thoroughly than in those with wide pupils.

"Whether, indeed," he concludes, "the lessened power of adaptation in the eyes with wide pupils is primary, and itself determines the size of the pupil, or whether, contrariwise, some weakness of the pupil-reflex, coming from another cause, constantly exposes the eye to light-stimuli of supernormal strength, and thereby weakens its adaptation—mechanism or chemism—this question remains unsolved."

He acknowledges that his observations have yielded "a picture peculiarly lacking in order."

ARTHUR J. BROCK.

OBSERVATIONS ON THE LIVING DEVELOPING NERVE FIBRE.

(189) ROSS G. HARRISON, *Amer. Journ. Anat.*, Vol. vii., No. 1, 1907, p. 116.

THE author, by a series of ingenious experiments, has been able to observe under the microscope the growth of the axis cylinder from a nerve cell. Portions of the spinal cord were taken from frog embryos shortly after the closure of the medullary folds, placed in a drop of frog lymph and observed on a hollow slide. The writer succeeded in keeping the nerve cells alive for nearly four weeks in some cases. The axis cylinder processes were exceedingly fine, almost hyaline in appearance, and devoid of the yolk granules which were numerous in the cell-bodies. A fine fibrillation was occasionally found in the protoplasm. The fibres were varicose, grew with an enlarged end, from which sprang numerous fine filaments. The enlarged end continually changed its form,

especially as regards the origin and the branching of the filaments. Its forward movement resulted in the drawing out and lengthening of the fibre to which it was attached, one fibre being observed to lengthen almost $20\ \mu$ in twenty-five minutes. The longest fibres observed were 0.2 mm. in length.

When portions of myotomes were left attached to a piece of the medullary cord, the muscle fibres after two or three days exhibited frequent contractions. The development of the axon is thus the result of amœboid movement of part of the nerve cell.

ALEXANDER BRUCE.

ACTION OF THE X-RAYS ON THE EYE IN COURSE OF DEVELOPMENT. (Action des rayons x sur l'œil en voie de développement.) TRIBONDEAU and BELLAY, *Arch. d'Élect. Méd.*, déc. 10, 1907 p. 907.

IN the course of their experiments on new-born cats, the authors have noted some changes already described in adult animals, such as various inflammatory affections of the conjunctiva, cornea and aqueous humor, with temporary loss of the eye-lashes. They have also found changes which are peculiar to young animals. These are: (1) earlier opening of the palpebral fissure on the exposed side, due to more active destruction of the cells in the epithelial septum which unites the eye-lids to each other; (2) delay in the normal pigmentation of the iris and nictitating membrane; (3) cataract, even with minimal doses; (4) microphthalmia, a constant phenomenon when the treatment is long continued; (5) microscopical malformation of the retina in the shape of a peculiar folding of its external layers, especially near the ora serrata; and (6) an occasional fibrillation of the vitreous humor, with a thickening of the hyaloid membrane.

These observations show that the rays cause injuries in young animals to organs which in adult animals can resist their action. They constitute a warning to electricians to be careful in treating diseases of the ocular region in children by the X-ray method.

ALEXANDER BRUCE.

A NEW PROOF OF THE CONDUCTING FUNCTION OF NEUROFIBRILS. (Ein neuer Beweis für die leitende Funktion der Neurofibrillen, nebst Bemerkungen über die Reflexzeit, Hemmungszeit und Latenzzeit des Muskels beim Blutegel.) A. BETHE, *Pflüger's Arch.*, March 1908, Bd. 122, p. 1.

THE author of this important contribution to the physiology of nerve starts with the assumption that in the Leech (*Hirudo*

medicinalis), where the natural elasticity seen in all nerve fibres to a lesser or greater extent is greatly exaggerated, the perifibrillar substance is elastic but the fibrils are not. He devotes some space to the proof of this, and reproduces some of Apathy's specimens of leeches fixed in conditions of physiological extension and contraction. These show that the nerve fibres are not thrown into wrinkles during contraction, but that they remain straight, the fibres being elastic; they also show that the fibrils within the fibres are thrown into wrinkles, the more marked the greater the contraction is, thus indicating that the fibrils retain their original length during the contraction of the leech though the fibres contract with the leech. He then applies this to the question of the conducting substance in the fibres: if that substance is the perifibrillar part of the fibre, then the conduction time of a given piece of nerve in the leech will be lessened if the animal contracts; on the other hand, if the fibrils be the conducting part of the system, then, as they do not shorten, the time of conduction will not be altered by contraction or elongation of the leech.

In the experimental part of the paper Dr Bethe describes his experiments to determine this question. He fixed a leech in the region of the uterus to a cork plate and registered the lengths of the front and hind segments, which were free, graphically. The stimulation was electrical and was applied near the hinder end, the latent-time of the anterior segment being measured. The stimuli were applied when the hinder end was in different states of contraction or relaxation and the latent times were measured. The latent time was found to remain constant within the limits of physiological stretching, but if these were exceeded then the latent time became longer.

As the fibrils remain constant in length within the limits of physiological stretching, he comes to the conclusion that they are the important factors in the conduction of the nerve-impulse, and that the perifibrillar substance has no influence on the rate of the impulse. He also finds that the reflex latent-time and the muscle latent-time depends on the tonic state of the reacting muscles, and that the latent-time of reflex inhibition is greater than that of reflex contraction.

T. GRAHAM BROWN.

**THE DEVELOPMENT OF THE ARTICULATORY CAPACITY
(192) FOR CONSONANTAL SOUNDS IN SCHOOL CHILDREN.**

ERNEST JONES, *Internat. Arch. für Schulhygiene*, Bd. iv., Heft 2, S. 186-201.

THE maximum articulatory capacity for 237 words was repeatedly tested in 450 normal school children, twenty-five of each sex for each year of school life, and marks allotted according to a

system here described. The 105,000 tests thus obtained are analysed from different points of view, and the results recorded in tabular form. The following conclusions are reached:—

A. *Relation to age.*—In both sexes progress in the perfection of this capacity is found principally at two years of school life; in boys these years are seven and eight, the former being rather the more important; in girls they are six and ten, the latter being rather the more important.

Less important steps are also found at other years. With boys, at the age of ten improvement is found to have occurred in the worst-speaking boys only; with girls, at the ages of seven and twelve, especially the former, improvement is found to have occurred in the better-speaking girls only.

B. *Comparison of the two sexes at different ages.*—On the whole the capacity is greater in the case of the girls, chiefly owing to the large number of boys in whom it is unusually badly developed; they excel in six of the nine years. In four of the years the difference between the sexes is negligibly slight. In the other five there is a decided difference between the capacity of the two sexes. At one of these years, that of nine, the boys excel; at the other four, namely at six, seven, twelve and thirteen, the girls excel.

The greatest sex difference is found at the age of six, and the next at the ages of seven and nine. AUTHOR'S ABSTRACT.

CLINICAL NEUROLOGY.

A CASE OF PERIPHERAL NEURITIS RESEMBLING TABES.

(193) (*Um caso de nervo-tabes peripherica.*) By H. D. ESTRADA,
Arch. Brasil. de Psychiatr., Neur. e Sciencias Affins., 1907, Nos.
3 and 4.

ON account of its supposed rarity the author records the clinical features of a patient in the Hospital de Misericordia at Rio. The patient, 32 years of age, syphilitic and potator, admitted with mitral and aortic valvular disease, was found to exhibit ataxia, Rombergism, absent knee-jerks, diminished heel-jerks, parasthesia and analgesia of the feet, and increased superficial reflexes. Pupil reactions, however, and vibratory sensibility were normal, there was pain on gastrocnemial pressure, lumbar puncture gave negative results, and the diagnosis of alcoholic polyneuritic pseudo-tabes was confirmed by subsequent rapid improvement under electrical and tonic treatment with massage, the disturbances of motility lasting barely one month.

R. CUNYNGHAM BROWN.

ANTERIOR PARÆSTHETIC MERALGIA. (Beitrag zur Kenntniss (194) der Meralgia paraesthetica anterior.) LASAREW, *Deutsche Zeitschr. f. Nervenheilk.*, Bd. xxxiv., S. 154.

SOME hundreds of cases are on record of the Roth-Bernhardt syndrome (external paræsthetic meralgia), but only three of anterior paræsthetic meralgia. Lasarew here reports a fourth case, which occurred in a woman of twenty-eight. The symptoms were typical and had been present for six years: paræsthesia and hypoæsthesia for all forms of sensation, and pain only on standing or walking, strictly confined to the territory of the middle cutaneous nerve. In the previous cases meralgia was present in the territory of both the external and middle cutaneous nerves, so that Lasarew's is the first case of pure anterior paræsthetic meralgia.

ERNEST JONES.

ON A CASE OF ACUTE POLIOMYELITIS ASSOCIATED WITH (195) A DIPLOCOCCAL INFECTION OF THE SPINAL SAC. PASTEUR, FOULERTON, and MACCORMACK, *Lancet*, Feb. 15, 1908, p. 484.

CLINICALLY this was a severe case of poliomyelitis in a boy of 13½, with involvement of almost the whole muscular system of the trunk as well as that of the limbs. But the chief interest lies in the fact that from the cerebro-spinal fluid obtained by lumbar puncture there was obtained a diplococcus of large size, the cocci being always free in the fluid. The cellular elements of the fluid were entirely lymphocytes. By inoculating some of the fluid in rabbits an ascending motor paralysis was produced after long incubation (about seven weeks). On the death of the experimental animal they recovered from its cerebro-spinal fluid a micrococcus similar to that from the human case. By inoculation of another rabbit with an emulsion of cerebro-spinal substance and fluid from the first experimental animal they again produced motor paralysis after a somewhat prolonged incubation period, and again associated with the presence of the micrococcus in the spinal fluid. Culture of the organism on artificial media failed entirely.

J. H. HARVEY PIRIE.

INFANTILE PARALYSIS. GUTHRIE RANKIN, *Practitioner*, Feb. (196) 1908, p. 166.

THIS is a good clinical lecture, but with no new facts to record.

J. H. HARVEY PIRIE.

**A CASE OF ACUTE ASCENDING PARALYSIS, WITH RE-
(197) COVERY.** C. W. VINING, *Lancet*, Feb. 8, 1908, p. 425.

THE case was that of a man of twenty-four who, in five days, developed complete paralysis of the legs, almost complete paralysis of the arms, the diaphragm, one side of the face and tongue muscles being also involved. Although commencing in the legs the parts affected were not altogether in succession from below upwards. The reflexes were entirely lost, there was some anæsthesia of the palate, and slight sensory disturbance on the legs for a few days, and incontinence of urine and fæces. A streptococcus was found in the cerebro-spinal fluid, but no growth could be obtained. Although not a classical case of Landry's paralysis, it corresponds better with that disease than any other; there were no evidences of diphtheria, syphilis, gonorrhœa, lead or alcohol. When the diaphragm became involved the case threatened to be rapidly fatal; strychnine was the sheet anchor in treatment, and appeared to be very beneficial. Recovery after a week of grave suspense was rapid and complete.

J. H. HARVEY PIRIE.

**LANDRY'S PARALYSIS; RECOVERY, PARTIAL RELAPSE AND
(198) COMPLETE RECOVERY.** JOHN K. MITCHELL, *Journ. Am. Med. Assoc.*, Feb. 1, 1908, p. 351.

A REPORT of an apparently typical case in a man of twenty-one with acute onset after a thorough wetting, rapid ascent of paralysis till all four limbs were involved, the reflexes being entirely abolished, sensibility normal and sphincters uninvolved. On the eighth day there was a distinct reaction of degeneration in anterior tibial and calf muscles of one leg. He was treated by galvanism to the spine, alternating applications of hot and cold water to the back, and small doses of ergot. In a little under three months he was discharged, walking well, though with still a small weakness in the legs. Four months later there was perhaps a slight relapse, which rapidly passed off, with complete recovery, but there is no exact record of the conditions during this stage, and it may quite well have been a passing nervous difficulty of an imaginary kind.

J. H. HARVEY PIRIE.

TABES A FEW YEARS AFTER INFECTION. (Ueber Tabes
(199) in den ersten Jahren nach der Infektion.) GALEWSKY,
Medizinische Klinik, Feb. 23, 1908, p. 260.

As a rule five to fifteen years elapse between syphilitic infection and the onset of tabes. Galewsky records six cases in which the symptoms of tabes occurred at a much earlier date. In two cases

they appeared in the second year. In the third case the symptoms of tabes made the patient first seek advice three years after infection, but they had certainly arisen a long time before. In the remainder they developed in the fourth or fifth year. In two of the cases syphilitic phenomena co-existed with tabes (*cf. Rev. of Neurol.*, 1906, p. 748, and 1907, p. 633). In one case syphilis showed a special malignancy, but in the others it was relatively benign. All the cases were alike in having had very little treatment or none at all.

Alcoholic and venereal excesses, neurasthenia, family predisposition, and the hardships of a campaign were among the causes that favoured the development of tabes in these cases.

J. D. ROLLESTON.

ON SEGMENTAL ABDOMINAL PARALYSES. (*Ueber segmentäre (200) Bauchmuskellähmungen.*) P. SALECKER, *Deutsche Zeitschr. f. Nervenheilk.*, 1908, Bd. xxxiv., Heft 2, S. 160.

THE writer comes to the following conclusions:—

1. There exist not only total, but also partial paralytic affections of the abdominal muscles in diseases of the spinal cord.
2. The innervation of the abdominal muscles is not multi-radicular but segmental.
3. The nuclei of the recti do not extend so far backward as those of the oblique muscles.
4. Individual abdominal reflexes appear to correspond to definite segments of the spinal cord.
5. Observations on segmental abdominal palsies, when taken together with sensory and reflex abnormalities, are of considerable diagnostic value for segmental localisation in the thoracic region of the cord.

Some four years ago Oppenheim (*Deutsche Zeitschr. f. Nervenheilk.*, 1903) maintained that the abdominal musculature is innervated as a whole and not in segments or according to spinal muscle or parts of muscle. During a laminectomy Oppenheim took an opportunity of stimulating single nerve-roots and produced a diffuse contraction of the corresponding abdominal muscle. He therefore concluded that the innervation of the abdominal muscle is multi-radicular, *i.e.* that every muscle is supplied by every nerve of the lower thoracic segments. Oppenheim also directed attention to the absence of clinical cases where localised atrophy was present in a segment of a rectus or obliquus muscle. But since then Ibrahim and Hermann (*Deutsche Zeitschr. f. Nervenheilk.*, 1905) have described four cases of poliomyelitis in which the oblique muscles were partially atrophied,

and one case of meningo-myelocoele where the oblique muscles were totally paralysed, the recti remaining powerful. Strasburger (*ibid.*, 1906) also described a case of poliomyelitis with total paralysis of the recti, the oblique muscles remaining unaffected.

Salecker here describes two cases of his own in which segmental paralysis was present in the abdominal muscles. One of the cases was completed by autopsy.

1. A previously healthy man, 40 years of age, was suddenly affected with unilateral shooting pains in the hip and leg of the right side; paræsthesia developed; then the opposite side became similarly affected; and finally there was produced a paresis of both lower limbs, rendering him unable to stand or walk. Clinical examination showed evidence of a lesion of the cord from about the 10th or 11th thoracic to the 3rd sacral segment. Anti-syphilitic treatment having failed, the diagnosis of extra-medullary tumour was made, as there was no dissociated anæsthesia or other sign pointing to an intra-medullary growth; the pain, etc., pointed to an extra-medullary lesion. The lower abdominal reflex was absent, the middle and upper abdominal reflexes were brisk. There was atrophy with reaction of degeneration in the lower third of the oblique muscles, whilst the recti and upper third of the oblique were normal. Operation revealed a glioma which unfortunately was intra-medullary and almost completely destroyed the lumbar enlargement. The patient died of pneumonia several months afterwards. Anatomical investigation showed that in the 1st lumbar segment a certain number of anterior cornual cells still survived (corresponding to the ileo-psoas muscles, which were unparalysed). The anterior cornua of the lowest thoracic segments were practically normal, but in the 12th, 11th, and partly in the 10th thoracic segments the anterior cornual cells were destroyed. Salecker therefore places the medullary centres for the lower third of the oblique muscles, together with the centre for the lower abdominal reflex, in the 12th and 11th thoracic segments. The recti in this case were intact, and their medullary centres, therefore, do not extend so far caudalwards as those for the oblique muscles.

2. A young and healthy workman had an injury in the lumbar region. This was followed by slight and transient sensory impairment in the cutaneous distribution of the lower thoracic and upper lumbar segments. Subsequently there developed muscular paralysis with reaction of degeneration. The adductors and quadriceps muscles were affected on both sides, with loss of the knee-jerks. In addition there was partial paralysis of the abdominal muscles—the lower third of the left rectus, the lower two-thirds of the left oblique, and about the lowest fourth of the right oblique muscle. Together with this there was loss of both

lower abdominal reflexes and of the middle abdominal reflex on the left side. The patient was completely cured in about four months. The diagnosis was that of a hæmorrhage in the cord extending from the 10th thoracic segment above to the 3rd or 4th lumbar segment below, the upper limit being higher on the left side.

PURVES STEWART.

**MENINGITIS IN MUMPS, WITH INVOLVEMENT OF THE
(201) FIFTH NERVE AND HERPES OF ONE OF ITS BRANCHES.**

(*Méningite lymphocytaire ourlienne, avec atteinte du trijumeau et zona d'une de ses branches.*) C. DOPTER, *Progrès médical*, 1908, p. 101.

A SOLDIER, who had already had mumps twelve years previously, had a second attack at twenty-two. Headache, vomiting, high temperature and a slow pulse supervened, and the diagnosis of meningitis which these symptoms indicated was confirmed by lumbar puncture. Three days later sensory troubles in the distribution of the fifth nerve occurred, consisting of hypoæsthesia in the region of the first division, hyperæsthesia in that of the second, and in that of the third hyperæsthesia was associated with the development of herpes. There was no orchitis, as in many of the cases of meningitis following mumps, nor was there any subsequent paralysis.

J. D. ROLLESTON.

MENINGITIS IN MUMPS. (Contribution à l'étude de la méningite

(202) ourlienne.) VENNAT, *Arch. de méd. et de pharm. militaires*, Feb. 1908, p. 143.

THE writer reviews the literature, and records a personal case in a soldier. All the characteristic symptoms of meningitis were present, especially headache, stiffness of the neck, and Kernig's sign, pyrexia and bradycardia (*cf. Rev. of Neur. and Psych.*, 1907, p. 301). Lumbar puncture could not be performed through want of a suitable syringe. Recovery took place. Vennat lays special stress on the beneficial effects of hot baths, which were given four-hourly.

J. D. ROLLESTON.

INTRACRANIAL ABSCESS DUE TO THE TYPHOID BACILLUS.

(203) GURD and NELLES, *Ann. of Surg.*, Jan. 1908.

A MACHINIST, aged 25, under treatment for typhoid fever, exhibited signs of a severe bruise on the right side of the head, above and in front of the ear, and extending forward to the supraorbital ridge. This was supposed to be the result of a blow sustained about a

month previously, but the history was somewhat vague. Redness, œdema, and fluctuation developed over the bruised area, and incision under chloroform revealed an abscess containing pus and blood, the evacuation of which exposed an area of bare bone with a lineal fracture running across it. The bone was trephined, and a large extra-dural clot partly organised and beginning to undergo suppuration was discovered. The patient made a good recovery. The typhoid bacillus was isolated from both the extra- and intracranial abscesses. The sequence of events in this case had been that, as a result of an injury, an extra-dural hæmorrhage had occurred, the abscess ensuing on account of blood-borne infection of the hæmatoma by the typhoid bacillus. HENRY J. DUNBAR.

HÆMORRHAGE INTO THE PONS VAROLII AS THE IMMEDIATE CAUSE OF DEATH IN THE ECLAMPSIA OF PREGNANCY, WITH ILLUSTRATIVE CASES. CARVER and FAIRBAIRN, *Med. Press*, Jan. 29, 1908, p. 116.

IN this communication the authors record a very interesting case, and review other cases illustrating the occurrence of hæmorrhages in fatal cases of eclampsia.

The patient was a primigravida, æt. 24. When about five months pregnant on the evening of 23rd March she complained of vague pains; her mental condition was good. She stated that for two or three days she had suffered from loss of appetite, sleeplessness, headache, and spots before the eyes. There had been neither vomiting nor fits. She had noticed no diminution in the amount of urine passed. On examination there was found to be general œdema of the whole body. The uterus reached the umbilicus, foetal heart sounds were audible. The temperature was normal, the pulse-rate 84, the arterial tension was considerably raised. A catheter specimen of urine became solid on boiling. Patient was ordered to remain in bed, a saline purge was administered, and arrangements were made to admit her to hospital next day. About 1.20 A.M. on 24th March (*i.e.* six hours after patient had been seen) patient's husband wakened and found his wife unconscious lying on her face and knees on the floor; before going to sleep he had noticed her hand twitching.

When seen at 2 A.M. the patient was cyanosed and quite unconscious, respirations were shallow and of the Cheyne-Stokes type, pulse 84, tension very high, all reflexes absent. No unilateral symptoms were noticed, there were no twitchings. The pupils were equal and moderately dilated. On arrival in hospital the pulse was found to be weaker and the respiration more markedly Cheyne-Stokes. Ten ounces of blood were withdrawn

from a vein and four pints of normal saline solution infused without any improvement of patient's condition. Rapid dilatation of the cervix was carried out and a living five months' foetus extracted by version, which only survived a few minutes. Patient's condition did not improve; she died at 7.15 without regaining consciousness.

Post-mortem examination showed that there had been an extensive hæmorrhage into the substance of the pons, which was greatly torn and disorganised. The hæmorrhage passed up for a short distance into the crura cerebri. The rest of the brain was normal. There was no disease of the cerebral arteries. Though the post-mortem shows death to have been due to a cerebral hæmorrhage, the authors maintain that it also affords evidence in support of their view that the case was primarily one of eclampsia. The renal changes were slight, but were definitely those of fatty degeneration and necrosis, such as are often found in eclampsia. The liver changes were not characteristic of those found in the toxæmia of pregnancy.

The symptoms of headache and sleeplessness first complained of point to a pre-eclamptic stage: there was no definite evidence of a fit except the twitching of the hands, and the further fact that she was found out of bed on the floor.

In regard to any alternative explanation the authors point out the difficulty of explaining a large hæmorrhage of this kind in a previously healthy young woman of 24 with no arterial lesions; they maintain that it is a fair presumption that the poison of the eclampsia was the determining cause of the apoplexy.

The authors proceed to discuss the incidence of cerebral hæmorrhage in eclampsia. Though definite brain changes are not found in eclampsia, it is not by any means uncommon to find small, usually meningeal, hæmorrhages which, however, are quite distinct from extensive hæmorrhages. Schanta's statistics show that in 90 post-mortems 10 cases showed evidence of cerebral hæmorrhage; in four it is described as "apoplexia gravis," in the others as "apoplexia capillaris." Schrieber in twenty-seven fatal cases notes no large cerebral apoplexy, but records four capillary hæmorrhages. Glockner in twenty-six examinations noted cerebral hæmorrhage in three, meningeal hæmorrhage in one; he does not give details as to the size of the hæmorrhages. The authors then minutely consider four cases of extensive hæmorrhage.

The first case they review is one recorded by Pfannenstiel (*Zentralb. f. Gynäk.*, 1897). This case has many points of interest. The patient, a primipara, æt. 22, in labour and unconscious, was admitted to his clinic at 9 A.M., as suffering from eclampsia. The history was that up to within a few days she had had perfect health; for a day or two her feet had been swollen. Four hours before admission she had an attack of sickness and a short fit, after which

consciousness had not returned. On admission the limbs were found to be limp; there was œdema up to the knees. Pulse was 68; respirations shallow, 44. The pupils, which were widely dilated, were of equal size; the corneæ were insensitive; the face was symmetrical. Sensibility was lost all over the body. The urine, which contained a few casts, became solid on boiling. In spite of active treatment, the patient died at 3.15 P.M.

Examination of the brain showed complete destruction of the left optic thalamus and surrounding parts by a hæmorrhage, which had broken through into the left lateral ventricle, with softening of the right optic eminence and filling of the ventricles with dark blood. There were points of blood in the pons. The arteries were normal except in the neighbourhood of the hæmorrhage. In the optic eminence was a varix the size of a cherry stone, with fresh thrombosis, and microscopically the position of the bursting of this thin-walled sac was recognised. No varices were recognised elsewhere in the body. Pfannenstiel considers the case to be one of true eclampsia on account of the highly albuminous urine, the œdema, and the premonitory symptoms, as well as the changes found in the liver and kidney. The thin-walled varix in the left optic eminence was probably pre-existent, and must be regarded as a *locus minoris resistentiæ*; this, when the eclamptic attack raised the blood-pressure, gave way.

The authors further cite cases recorded by Meyer-Wiry, Maygrier, and Chavanc and Esch. In all four cases, however, the hæmorrhage was not actually into the pons. All the cases cited by the authors have one feature in common, viz., in none was the cerebral hæmorrhage recognised before death. Among other points of similarity it is important to note that all the patients were young primiparæ, the ages being 22, 24, 25, 25, 38. Cerebral hæmorrhages at such early ages point in a most striking manner to the grave effect of the eclamptic toxin on the arterial walls.

In all five cases there were some premonitory symptoms such as headache, ocular disturbances, twitchings or respiratory difficulties. The onset of coma was as a rule the first indication of the serious nature of the case. In some of the cases a fit preceded the coma, but deep coma supervened with little in the way of antecedent convulsions.

The breathing in all cases was early stertorous, and later of the Cheyne-Stokes type. Cyanosis was a marked feature of all cases. Though the amount of dilatation varied in different patients, the pupils in all cases were dilated, equal in size, and inactive.

MALCOLM CAMPBELL.

THE MUSCULAR STRENGTH OF HEMIPLEGICS. (Über die
(205) Kraft der Hemiplegiker.) MAX STERNBERG, *Deutsche
Zeitschr. f. Nervenheilk.*, Bd. xxxiv., S. 128.

STERNBERG reports the results of careful and repeated tests, graphically recorded, made on twenty-seven hemiplegics. The object of the research was to check Pitres' findings, that the healthy side shows some loss of power, and that simultaneously-made bilateral efforts increased the strength exerted by the paralysed limbs. The first of these statements was fully confirmed, the second not at all. According to Broadbent's theory, bilateral effort should diminish the power of the healthy side and increase that of the paresed. Although in some cases this was true, in others it was not. Sternberg finds that the effect of bilateral effort on the paresed limbs may be either to increase or diminish their power, and that this effect may vary in different tests; similarly on the healthy side. He makes some important remarks on the technique of dynamometer testing, for which he uses a new instrument he has invented (see *Neurologisches Centralblatt*, 1907, No. 11), and on the bearing of the present results on the theory of the recovery of power in hemiplegia.

ERNEST JONES.

PSEUDO-HYSTERICAL HEMIPLEGIA. (Hemiplegia pseudo-
(206) hysterica.) ADAMKIEWICZ, *Neurol. Centralbl.*, Feb. 1, 1908,
S. 98.

THE author maintains that as hysterical hemiplegia may resemble that of organic origin, so may the latter sometimes present the features characteristic of the former. In illustration of this he reports the case of a woman, aged 50, who, on receipt of some distressing news, became excited and suddenly paralysed on the left side. The hemiplegia was complete, and she was unable to make the slightest movement with the paralysed limbs. The cranial nerves were, however, all normal. Complete hemianæsthesia, limited by the middle line, and not implicating the face, existed for all forms of sensation. No note is made of the plantar reflex.

In favour of the diagnosis of hysterical hemiplegia was the mode of onset, the intactness of consciousness and intelligence throughout, the complete hemianæsthesia, the absence of hemianopia and of paralysis of the face or tongue, and the clearness of speech. Against this, however, was the absence of hemianosmia, hemiægeusia, and hemianopia. The author uses the last argument first, to exclude the hysterical nature of the affection and then to exclude the organic nature of it, though few authorities would maintain that hemianopia is found with hysterical hemiplegia as often as with organic.

On the basis merely of absence of hemianosmia and hemiageusia, the author inclined to the diagnosis of organic hemiplegia, though it might be remarked that one would not expect to find these symptoms in a case in which both the motor and sensory functions of the face were normal, as here. To decide the problem he resorts to what he calls the crucial test of the application of mustard paste to the anæsthetic side, for he holds that this invariably determines the transference of hysterical hemianæsthesia to the opposite side. As in this instance the test was negative, his original diagnosis was confirmed.

It comes as a surprise to read such a contribution in the pages of the *Neurologisches Centralblatt*.
ERNEST JONES.

**THE FUNCTIONS OF THE CORPORA STRIATA, WITH A SUG-
(207) GESTION AS TO A CLINICAL METHOD OF STUDYING
THEM.** C. L. DANA (of New York), *Journ. Nerv. and Ment.*
Dis., Feb. 1908.

THE paper discusses in a most instructive way the anatomical structure and the physiology of the striate body, citing the clinical and pathological results of experimental work done by different investigators. Four cases are cited in which a careful clinical study had been made and autopsy performed. The author refers briefly to the clinical manifestations and post-mortem findings in the lenticular nucleus of gas-poisoning cases. He summarises his work as follows:—

The corpus striatum has not any independent or specific motor function. It probably has some supplementary motor function, especially in connection with articulation. It may have some control over the bladder (double lesions). It seems to have some control over vaso-motor and trophic conditions of the skin (and lungs?). It has no thermic centre. It may have some supplementary and associative psychic function, so that lesions affect memory or initiative. It is an organ of less importance relatively in the higher vertebrates. In severe gas poisoning there is a double softening of the lenticular nuclei, due to thrombosis of "the artery of cerebral thrombosis," and there result vaso-motor and gangrenous conditions of the skin, so that these conditions in connection with a history of coma from gas poisoning form a group of symptoms called "the syndrome of the corpus striatum."

C. H. HOLMES.

**CLINICAL CONTRIBUTIONS TO THE QUESTION OF ACUTE
(208) TOXIC AND INFECTIOUS NEURITIS OF THE AUDITORY
NERVE.** (Klinische Beiträge zur Frage der akuten toxischen
und infektiösen Neuritis des Nervus Acusticus.) J. HEGENER,
Zeitschr. f. Ohrenheilk., Bd. 55, H. 1-2, 1908, S. 92.

THE author contrasts the advantages possessed by the ophthalmologists for observing the optic nerve during life and testing its condition with the difficulties attending the examination of the auditory nerve during life, and even after death. Siebenmann has described cases of interstitial auditory neuritis in cancerous cachexia and of retrolabyrinthine neuritis in pulmonary tuberculosis, but Wittmaack must be given the credit of recognising a toxic auditory neuritis analogous to optic neuritis. He found that in animals injected with tubercle as well as in men the cochlear branch of the nerve is the one that is first and most severely affected: this explains the prominence of deafness in these cases, and the almost complete absence of giddiness. Clinically such cases are characterised by a diminution of hearing, gradually increasing as one passes up to the upper tone limit, combined with lowering of this limit. This is analogous to the toxic affection of the central bundle of the optic nerve. Gradenigo thinks that damage of the middle of the field of hearing is characteristic of toxic affections, but Hegener agrees with Wittmaack.

Manasse has investigated post-mortem material from old people who had become deaf, and finds that the ramus cochlearis is usually diseased, less frequently the cochlear and vestibular branches together; and most rarely the vestibular division was more severely and therefore primarily affected.

Hegener gives details of six cases. Case I.: M., aged 22, caught cold on a long journey. Later diarrhoea and a feeling of sickness came on, followed by giddiness, left-sided facial paralysis, and indistinctness of vision on looking to the left. Two weeks later deafness came on in the left ear, but there was no tinnitus, ear-ache, headache or anæsthesia of the face: dysphagia, difficulty in speaking, and disturbance of taste were also absent. The patient swayed a little when standing with eyes closed, and tended to fall to the left. When asked to walk straight forward he described a semicircle to the left, but exhibited no nystagmus or facial spasm. He stood the faradic current well, but galvanism produced extreme giddiness with tendency to fall to the left. Examination of the ear showed that the middle ear was normal. The tuning fork on the vertex was lateralized to the healthy ear, and both bone and air conduction were shortened on the left side—air conduction being the longer. The lower tone limit was 24 V.D. and the

upper tone limit for the tuning fork 1024 V.D., but the Galton-Edelmann whistle was heard up to 17,000 V.D.

The patient was treated with aspirin, rest, and faradic current. The facial paralysis rapidly improved, the upper tone limit of the left ear rose to 20,000 V.D. for the whistle, and C. 5 (4096 V.D.) could be heard when forcibly vibrated. The second examination of the hearing showed that certain tones about the middle of the scale were heard better than normal, and Hegener suggests that this may have been due to the stapedius paralysis. Eventually the case completely recovered. Case II.: M., 31., was probably of rheumatic origin. Tinnitus was first complained of, followed by deafness, and later on by giddiness, nausea, and vomiting. After three days in bed the vomiting disappeared, the giddiness got less, but the deafness and tinnitus still remained. In this case middle ear catarrh was also present, and the lowest tones were not heard. The upper tone limit as tested by the whistle was 14,000 V. D. Slight nystagmus was present, but otherwise the vestibular apparatus was not affected. Case III.: M., aged 40, was due to influenza. Tinnitus and deafness came on four days after the beginning of the illness, but there was no giddiness. Salipyrin made the tinnitus much worse—an instance of a synthetic poison acting on an organ already diseased. After two years the hearing had only returned for tones in the middle of the scale. Case IV. was similar, but the patient was sixty-five years old. The other two cases were diabetics. In the first the upper tone limit was much reduced, but in the second the lower tones were not heard, though the bone conduction was shortened. Siebenmann says that infectious diseases tend to produce nerve changes in an ear already affected by stapes ankylosis, and Scheibe has described an acute unilateral affection of the 8th nerve characterised by deafness for the lowest tones. Hegener compares these pictures of limitation of the hearing field to cases of optic neuritis with scotoma. The paper is very interesting, if not absolutely convincing, and the author rightly emphasizes the necessity of the aurist working in conjunction with the physician.

J. S. FRASER.

A CASE OF ACROMEGALY WITH OSTEO-ARTHROPATHIES

(209) **AND PARAPLEGIA.** (*Sur un cas d'acromégalie avec ostéo-arthropathies et paraplégie.*) By V. BEDUSCHI (Milan), *Nouv. Icon. de la Salpêtrière*, No. 6, 1907.

IN this short paper Dr Beduschi records the clinical features of a case, a woman æt. 30 years, who, in addition to the characteristic appearances of acromegaly, presented flaccid paralysis of both legs and grave osteo-articular dystrophies. The principal interest

centred round the results of the examination of the nervous system. Visual fields, pupils, external ocular movements, and all special senses were normal, but in the upper extremities the deep reflexes were diminished and in the lower limbs abolished. Further, there was paralysis with atrophy of the flexors and extensors of hip and knee on both sides, with in-excitability, or in some muscle groups hypo-excitability, to faradic stimulation.

Proceeding to the discussion of the exostoses—the text being illustrated by radiographs—the author separates these from the arthropathies of tabes and syringomyelia, and relates the whole clinical appearances to the single and as yet unascertained cause of the acromegaly, *i.e.* to the other distinctive forms of acromegaly is to be added another, a paralytic form.

R. CUNYNGHAM BROWN.

TEMPORARY OEDEMA OF ONE OPTIC DISC AS A LOCALISED (210) MANIFESTATION OF ACUTE CIRCUMSCRIBED (ANGIO-NEUROTIC) OEDEMA (Quincke). (*Kurzdauerndes Oedem der Sehnervenpapille eines Auges, eine Localization des akuten umschriebenen Oedems* (Quincke). HANDWERCK, *Münch. med. Wchschr.*, No. xlviii., 1907, p. 2332.

HANDWERCK describes a case of an old woman of 73 in whom there had been at intervals various manifestations of acute circumscribed oedema. In mid-September she had shown several manifestations of this disease; during the winter she had been fairly free from attacks; but in the middle of April she had two attacks in succession, one area being on the chin, the other on the under lip. On the 19th April the left side of her tongue was definitely affected. On the 20th April she complained of a sensation of white flickering before her eyes, like white leaves falling to the ground, mostly affecting her right eye. On the 22nd April an ophthalmoscopic examination was made. Her visual acuity was good, her visual fields were full, her pupil reactions normal, conjunctiva healthy; she had hypermetropia + 2.5 D. The right optic disc showed blurring of the edges, with hyperæmia and 2 D. of swelling. The fovea was also hyperæmic, arteries were diminished, and the veins dilated.

In the course of eight days the whole process had subsided, leaving the right fundus normal. The author ascribes this passing change to a local manifestation of angioneurotic oedema.

LESLIE PATON.

ALCOHOLIC EPILEPSY. (*L'Epilepsie alcoolique.*) L. ELIÉ,
(211) *Journ. de Méd.*, Jan. 25, 1908.

It is necessary to distinguish between epileptic attacks occurring in the course of subacute alcoholism and epileptiform convulsions met with at an advanced period of chronic alcoholism.

Acute Alcoholism.—These attacks occur usually in the case of an habitual drinker who has passed the point of saturation. Such a case has visual and auditory hallucinations, and does not know where he is. The period of occurrence of the epileptic attack is not fixed, and it cannot be foreseen. Some patients have an attack in their first subacute alcoholic delirium, others not till their second or third. In the writer's experience the seizures have been intense in character, the violent movements of the arms and legs suggesting a comparison with hystero-epilepsy. Aura and cry are absent. The face of the idiopathic epileptic is pale, that of the alcoholic subject congested and the eyes injected, except in the case of absinthe drinkers, when pallor is the rule. The epileptic does not recollect his seizure or the delirium which may follow it. The alcoholic may, on the other hand, retain the memory of his delirium. It may be possible by strong effort to fix the attention of the alcoholic and obtain a certain response, but this could with difficulty be done in an epileptic.

The seizures may be single or repeated several times. When the patient abstains from drink they diminish in frequency, and rapidly disappear when the toxic effects of alcohol are got rid of. They reappear again if indulgence be resumed.

Prognosis is essentially favourable. In fatal cases at the autopsy a simple generalized congestion of the meninges and the brain has been found.

As to diagnosis, only the progress of the case and the effect of the withdrawal of alcohol enable one to say whether one is dealing with a case of alcoholic epilepsy or with an epileptic whose seizure has been coincident.

Chronic Alcoholism.—Alcohol can eventually bring on convulsions, but these do not appear in the form of a frank epileptic attack, but as epileptiform seizures, analogous to those met with in cases of general paralysis and brain tumour. They occur in those who have long shown the signs of chronic alcoholism. They are not fixed in occurrence. There is no aura, though sometimes they are preceded by a dulness in the head and general malaise. The convulsions which characterise them are not general, but are limited to one side of the body or face, or one of the limbs. They may predominate in one group of muscles. They consist in clonic movements of a part or in involuntary or rapid movements of deglutition. From these characters, and their tendency to self-limitation, they resemble the choreiform movements of general

paralytics or patients affected with cerebral tumour. The patient remains more or less aware of what is going on around him and does not lose consciousness. He is feeble, his face congested, the temperature rising to 38·5° or 39°. Recovery is rare.

A. HILL BUCHAN.

POST-DELIRIOUS ALCOHOLIC STUPOR (Alcoholic Cerebral Oedema, (212) "Wet Brain"). CLARLES K. STILLMAN, M.D., *New York Med. Journ.*, Jan. 25, 1908.

THIS peculiar stupor has been little studied. The association of stupor with oedema of the meninges has led many to regard the transudate as the central factor in the condition; but it seems that the symptoms are merely those of a type of cerebral irritability. For they may occur in the absence of oedema, *e.g.* in typhoid and uræmia; while on the other hand serous effusion may be found in cases that during life do not manifest symptoms of the kind. The cause of the oedema has probably to be sought among the general conditions underlying the stupor. While it is true that what is generally spoken of as "alcoholic wet brain" is a transudate pure and simple, there is in alcoholic oedema a tendency to low-grade inflammatory changes about the blood vessels and meninges more marked than in "wet brain" of other types. In true oedema the effusion occurs well distributed, whereas in post-mortem oedema the fluid is found only in dependent areas. The special symptoms are almost always preceded by some alcoholic delirium. A study was made of 98 cases in the alcoholic wards of Bellevue Hospital. Out of 2133 patients admitted to the female alcoholic ward from September 1, 1905, to September 1, 1906, approximately 1 per cent. were attacked with "wet brain." The youngest was twenty-three years old, the oldest sixty; majority thirty to forty. About 81 per cent. died. Duration of illness in fatal uncomplicated cases varied from one to forty-five days. Of the non-fatal cases some were dismissed "improved." One woman, thirty-four years old, who suffered from the most aggravated form of attack, is now quite strong and healthy. During the same period there were 5017 admissions to the male alcoholic ward: 1·5 per cent. of them suffered from the condition in question—oldest seventy, 3 between sixty and seventy, 14 between fifty and sixty, 23 between thirty and forty, 4 between twenty-two and thirty. Mortality, 79 per cent. Duration in uncomplicated cases, from less than one to twenty-two days. Thirteen patients recovered in from two to forty-five days.

Men survive the shorter attacks better than women, but succumb more frequently to the longer ones. Men often have debauches in the course of steady tipping, which precipitate

attacks of delirium tremens, whereas women tipple steadily, but debauch less, and when stupor comes on, their vital powers are apt to be very low. In men one often sees what may be called "transitory wet brain."

The statistics show a tendency towards a separation into a short and a protracted type of attack. Wet brain oftenest comes on about the third or fourth day of delirium tremens. As regards symptomatology, one notes the alteration in facial expression: grey pallor and almost cadaveric immobility of countenance, the hands reaching up towards the head, which is thrown back; the pupils at first often pin-point, later dilated; the very persistent "stiff neck"; the pulse small, frequent, and of low tension; the temperature typically low—99-101 F. or subnormal. The degree of coma varies.

Diagnosis is often more difficult than might be supposed. Perhaps the most fruitful source of error is tubercular meningitis in adults. Possibly the best treatment is forced feeding with judicious stimulation. Enormous amounts of food are required. The patients do not react to ordinary doses of stimulants. Caffein is perhaps the best drug, given in five-grain doses of the citrate, or sodium benzoate. Sitting the patients up in bed is said to be attended with good results. Tapping the cord produces only temporary relief. The fluid removed shows no difference from normal fluid except that it is usually increased in amount and is under greater pressure.

A. HILL BUCHAN.

COMPLICATIONS OF ALCOHOLISM, WITH SOME STATISTICS

(213) ON TWO THOUSAND CASES. LEONARD D. FRESCOLN, M.D., *Journ. Amer. Med. Ass.*, Feb. 8, 1908.

THE complications and sequelæ of the continued use of alcohol are more to be feared than alcoholism itself.

An examination of 4000 cases of alcoholism at the Philadelphia General Hospital during the past three or four years showed a great variety of recorded "complications." A number of these were merely accidentally associated troubles, but there were a number of diseases running hand in hand with alcoholism in many cases.

In 2000 cases the "complications" which seemed most reasonably linked with alcoholism were as follows:—

Delirium tremens (7 deaths)	.	.	49 cases
Forms of nephritis	.	.	28 "
Pneumonia	.	.	23 "
Pulmonary tuberculosis	.	.	23 "
Myocarditis	.	.	20 "
Uremia	.	.	16 "

Bronchitis	15 cases
Gastroenteritis	14 „
Lacerations	14 „
Valvular heart disease	11 „
Contusions	10 „
Leg ulcers	9 „
Rheumatism	7 „
Pulmonary oedema	7 „
Fractures	7 „
Typhoid	6 „
Confusional insanity	6 „
Multiple neuritis	6 „
Erysipelas	5 „
Cirrhosis of liver	5 „
Epilepsy	4 „

Something must be allowed for want of sufficient care in case-taking; *e.g.*, arteriosclerosis was noted only in from 8 to 10 per cent., whereas it must be present in from 80 to 90 per cent. of cases.

For the most part those who drink to excess are the uncleanly, the poor, the ignorant, and especially those of bad heredity. The habits of the alcoholic explain many of the complications. The author has never seen a lobar pneumonia in a fat man suffering from delirium tremens end in recovery, but in lean subjects, several.

A. HILL BUCHAN.

ON THE DIFFERENTIAL DIAGNOSTIC VALUE OF WASSER-

(214) **MANN'S SERUM DIAGNOSIS OF SYPHILIS FOR INTERNAL MEDICINE AND NEUROLOGY.** (Ueber den differentiell-diagnostischen Wert der Wassermannschen Serodiagnostik bei Lues für die innere Medizin und die Neurologie.) K. KRONER (of Berlin), *Berl. klin. Wochenschr.*, Jan. 27, 1908.

CLINICAL CONSIDERATIONS ON WASSERMANN'S REACTION

(215) **IN SYPHILIS.** (Klinische Betrachtungen über die Wassermannsche Reaktion bei Syphilis.) W. FISCHER (of Berlin), *Berl. klin. Wochenschr.*, Jan. 27, 1908.

KRONER records the results of the examination of forty patients with regard to the reaction of their serum or cerebro-spinal fluid by Wassermann's method. The cases were divided into non-syphilitic (6), probably non-syphilitic (5), and syphilitic (30); in the third group doubt was excluded either by the anamnesis, the nature of the clinical symptoms, or the autopsy findings. In the first two groups the reaction was negative; out of the third group twenty-two gave a positive reaction.

A negative reaction, therefore, does not exclude previous infection, and, according to the author, a positive reaction may be given in certain cases of jaundice; this, however, does not materially alter the practical significance of a positive reaction.

Fischer, from a dermatological service, tested the reaction in 250 syphilitic patients, chiefly in early stages of the disease; in 84 per cent. the reaction was positive. It is doubtful whether a positive reaction can be expected before the seventh or eighth week after infection. The reaction allows of the diagnosis of the constitutional malady, but does not warrant the conclusion that any definite organ shows syphilitic changes; thus the pleuritic exudate in a patient, whose secondaries had disappeared less than two months previously, gave a positive reaction.

A negative result is not of diagnostic value; the reaction cannot be used to test the efficacy of therapeutic measures.

C. MACFIE CAMPBELL.

ON WASSERMANN'S SERUM DIAGNOSIS OF SYPHILIS. (Ueber (216) die Wassermannsche Serodiagnostik der Syphilis.) E. FRAENKEL and H. MUCH (of Hamburg). *Münch. med. Wochenschr.*, March 24th, 1908.

THE authors examined by Wassermann's method the serum of patients with certain diseases held to be related etiologically to syphilis—aortitis, orchitis, stricture of the rectum. In all the cases of aortitis the reaction was positive; in two cases of aortic aneurysm the result was negative. In the four cases of disease of the testicle examined the result was negative. In two cases of general amyloid disease, which, both on clinical and pathological examination, presented no evidence of any of the disorders usually associated with this condition, the result was negative: the spleen in both cases presented the ham-like appearance frequently considered to be pathognomonic of previous syphilis. The authors mention the discovery at the autopsy of definite syphilitic lesions in a patient who died from pneumonia. In such cases a positive reaction might be used wrongly as an argument to show that the Wassermann reaction is present in other diseases than syphilis. The importance of corroboration by post-mortem examination is emphasized.

C. MACFIE CAMPBELL.

NERVOUS MANIFESTATIONS OF ARTERIOSCLEROSIS. A. (217) STENGEL, *Amer. Journ. Med. Sc.*, Feb. 1908.

THE author emphasizes the distinction between symptoms that result directly from arteriosclerosis and those which have some intermediate organic derangement for their cause. He discusses

and gives cases illustrating symptoms associated with obstruction of peripheral vessels, painful sensations preceding senile and diabetic gangrene, paræsthesia of various kinds, pains associated with arteriosclerosis of the abdominal aorta and of its branches, and of the thoracic aorta, cerebral manifestations of arteriosclerosis.

C. MACFIE CAMPBELL.

A NEW CASE OF ANKLE CLONUS IN HYSTERIA. (Un
(218) nouveau cas de clonus du pied dans l'hystérie.) VAN
GEHUCHTEN, *Le Névrose*, Jan. 25, 1908, Vol. ix.

VAN GEHUCHTEN, who has done excellent work in breaking down the inviolability of the French dogmas concerning the non-occurrence of certain reflexes in hysteria, begins this article by refuting Collier's objection to a previously published case, namely, that it was one of early disseminated sclerosis. This case, eighteen months later, still presents not a single sign of disease of the nervous system, and it is to be hoped that in a couple of years the author will again publish a report of it. Collier's argument as to the limited evidence in favour of the thesis he answers by pointing out that the occurrence of Babinski's sign and ankle clonus in hysteria is acknowledged, if it occurs at all, to be very rare.

He now reports a second case, that of a woman aged thirty-five, who, after one of a long series of severe hysterical crises, was seized with a spastic paralysis of the left leg and a flaccid paralysis of the left arm. Four months previously she had lost hearing, sight, taste, and smell on the left side. On examination the paralysis and contracture were found to present typical hysterical features, and yielded to psychotherapeutic measures. Further, there was loss of sight, smell, and taste, and almost complete loss of hearing on the left side; the left side, including the mucous membranes, was completely anæsthetic. The right knee-jerk was normal, the left greatly exaggerated. On the left side a typical permanent ankle clonus was obtained, showing none of the features of false ankle clonus. The author, therefore, still maintains that exaggeration of the knee-jerk and true ankle clonus may be due to hysteria.

ERNEST JONES.

NOTE ON A CASE OF SPONTANEOUS SOMNAMBULISM.

(219) WARREN LLOYD, *Journ. of Abnorm. Psychol.*, Feb. 1908, Vol. ii., pp. 239-259.

THIS case is reported from notes made by the author when a psychology student over ten years ago. As the case was never scientifically investigated, it is of little or no value. All that seems

certain is that the patient, then a man of twenty-three, had spent a great part of his life, certainly since the age of twelve, oscillating between two states of consciousness. In the assumedly abnormal one he presented marked changes of character, notably irritability. Consciousness in this state included all previous memories, whereas that in the waking normal state included only memories of the same state. The case developed after a series of epileptiform fits, and seems to be an ordinary one of hysteric dual consciousness.

ERNEST JONES.

UNUSUAL ILLUSIONS OCCURRING IN PSYCHOLEPTIC ATTACKS OF HYSTERICAL ORIGIN. HARVEY CARR, *Journ. of Abnorm. Psychol.*, Feb. 1908, Vol. ii., p. 260.

CARR relates an interesting case in which attacks of the following nature occurred. The two features were: (1) a sense of all surrounding objects rapidly receding to the horizon; this applied also to the patient's own body. It usually culminated in not only blindness, but a sense of blankness in which nothing in the world seemed to exist; (2) a feeling of total paralysis and helplessness, evidently of an aboulie nature; this added to the unpleasantness of the experience. No other disturbances accompanied the attacks, which dated from the age of six. Fluctuating cutaneous anæsthesia, incessant nightmares, marked tendency to reverie were features that indicate the hysterical nature of the case.

Although a good descriptive account of the attacks is given, no psychological analysis of the underlying phenomena is mentioned. Particularly noteworthy is the lack of attention paid to the dreams, although the author repeatedly insists on their vividness, prominence, and the facility with which they could be recalled. Freud's epoch-making work on dream analysis as the most important method of studying sub-conscious feeling memories seems to be little known in America as yet.

ERNEST JONES.

THE VALUE OF CYTODIAGNOSIS IN PRACTICAL MEDICINE. (221) JAMES E. H. SAWYER, *Lancet*, Feb. 1, 1908, p. 283.

A SHORT paper dealing with the importance of examining serous effusions and cerebro-spinal fluid for lymphocytes, polymorphs, and endothelial cells as an aid in diagnosing tubercular, acute, inflammatory, or malignant diseases and mechanical effusions. Nothing new.

J. H. HARVEY PIRIE.

**RECENT RESEARCHES ON THE DIAGNOSTIC MEANING OF
(222) PUPILLARY SYMPTOMS.** (Neuere Untersuchungen über
die diagnostische Bedeutung der Pupillensymptome.) BUMKE,
Münch. med. Wchnschr., No. xlvii., 1907, p. 2313.

THIS paper, a contribution to a discussion, gives an interesting account of much recent work on the importance of variations of the pupillary reflexes in certain diseases of the central nervous system. Dealing first with the Argyll Robertson pupil, he points out that its existence is to be regarded as absolutely pathognomic of the metasymphilitic affections, tabes and general paralysis, though it has been shown that Argyll Robertson pupils may be present for years (as long as ten years according to Thomsen) before other signs of tabes or general paralysis become manifest. No one has abolition of the light reflex who is not syphilitic. He regards this as the most important symptom in the diagnosis, of more importance than any alterations in the leucocytes in the cerebro-spinal fluid, though he admits that such changes are relatively seldom missing and the Argyll Robertson pupil in a pure form may be relatively frequently absent. According to the older statistics 64 per cent. of tabetics and 62 per cent. of patients with G. P. I. showed definite Argyll Robertson pupils, 20-25 per cent. showed some alteration in the light reaction, and in only 15-20 per cent. was a diagnosis made independently of any change of the iris movements.

An advance in the refinement of methods has altered these percentages, but we are still without satisfactory means of standardising the amount of stimulation, and so measuring the minimal stimulus requisite to produce a reaction. Schlesinger's apparatus the writer regards as unsatisfactory. Weiler's method of noting the secondary reaction in an exposed eye on uncovering the opposite eye is regarded with more favour. This secondary contraction is said to be absent in 96 per cent. of general paralytics.

Bumke's own method depends on the fact that a normal relationship exists between the amount of a galvanic current necessary to give rise to a sensation of light and that necessary to cause any pupillary reaction. He finds that while .02 to .2 milliampères current gives rise to a sensation of light, pupil activity is called forth by a current between .04 and .5. The proportion between the two strengths is fairly constant in healthy people. In only 13 per cent. of general paralytics was any reaction produced by currents under 3 milliampères.

He does not agree with Wolf and Gaupp's assertion that Argyll Robertson pupils are distinctive of tabes as apart from G. P. I.

With regard to other pupillary disturbances, myosis is more frequent in tabes than in G. P. I. Absolute paralysis of pupils is rare in tabes, and certainly rarer than in dementia paralytica and cerebral syphilis. Ophthalmoplegia interna occurs occasionally in tabetics. Uhthoff saw it in five out of a hundred cases. When it is double-sided, in most cases it is probably not of syphilitic origin. Only eight out of thirty cases seen by Uhthoff were syphilitic.

Bumke does not regard the absence of reflex widening of the pupil in response to sensory stimuli as a symptom of any great value. In discussing the pupillary disturbances in functional diseases, he says that 69 per cent. of the cases grouped together under the heading of dementia præcox show no widening of the pupil in response to sensory or psychical stimuli. The pupils of such patients are, as a rule, rather wider than those of healthy people of the same age.

In epilepsy the pupil is fixed during the fits (Romberg), but Karplus and Westphal have shown that this fixity of the pupil may also occur in hysterical fits. The fixity may be in a position of partial dilatation or of contraction. Occasionally they have been described as being elliptical. In conclusion the writer discusses the occurrence of sympathetic paresis, and records the occurrence of a case of unilateral A. R. pupil with myosis following injury to the skull.

LESLIE PATON.

PSYCHIATRY.

FIVE CASES OF GENERAL PARALYSIS IN CHILDREN. (Über (223) fünf Fälle von progressiver Paralyse bei Kindern.) F. BACHMANN, *Fortschritte der Med.*, Jan. 20, 1908, p. 33.

Bachmann has recently observed five cases. Syphilis played the chief part in the etiology. In some it was inherited, in other cases it was possibly acquired. The mothers of two of the children had tabes. The children up to a certain age showed a normal intellectual and physical development, and then suddenly became backward at school. Their psychical condition offered the variety met with in the general paralysis of adults.

Besides the intellectual changes, all showed amnesia for recent events and alterations in their dispositions and emotions (lachrymosity and violent passion). There were no delusions or expansive ideas. The somatic phenomena were as varied as the psychical. All exhibited a disturbance of gait, which was clumsy or definitely ataxic, and affection of speech. Their writing re-

vealed a tremor and frequent omission of words or syllables. In most there was complete immobility of the pupils. On ophthalmoscopic examination the papillæ were found either normal or atrophic. In some the tendon reflexes were exaggerated, in others they were abolished. Sensibility in all cases was apparently intact. In only one case were there sphincter troubles. Lumbar puncture was performed in all. In three cases lymphocytosis was found. In one case it was impossible to obtain a specimen free from blood. Herpes zoster followed shortly after lumbar puncture in this case. In some of the children there were typical paralytic attacks, followed in most cases by deep sleep. One case had a remission lasting three months, during which period there were no paralytic attacks, and the child became quieter, displayed more interest in its surroundings, began to read again, and showed a distinct improvement in memory.

J. D. ROLLESTON.

ONSET OF GENERAL PARALYSIS THREE YEARS AFTER (224) SYPHILITIC INFECTION. (Cas de paralysie générale ayant débuté 3 ans après l'infection syphilitique.) EHLERS, *Bull. de la Soc. franç. de derm. et de syph.*, Jan. 1908, p. 24.

ONSET OF GENERAL PARALYSIS IN THE THIRD YEAR OF (225) SYPHILIS. (Paralysie générale débutant à la troisième année d'une syphilis.) FOURNIER, *Ibidem*, Feb. 1908, p. 48.

EHLERS' case was that of a man who contracted syphilis in June 1903, for which he underwent a short course of mercurial inunction. The secondary symptoms were mild. In August 1906 he was admitted to an asylum with general paralysis, the first symptoms of which had developed two months previously. In addition to the characteristic mental and physical signs of general paralysis he presented a generalised indolent adenopathy and a papulo-squamous eruption.

Fournier's case developed syphilis in 1880. The secondary symptoms were few and slight. Treatment was continued for only a few months. In a little less than three years after the chancre general paralysis appeared, and ran its course in less than two years, the patient dying at the age of twenty-four, in the fifth year of his syphilis. According to Fournier general paralysis is absolutely unknown during the first two years of syphilis, and only begins to appear in the third, a time when cerebral syphilis is most frequent. The most usual date for its development is comprised between the sixth and twelfth years.

J. D. ROLLESTON.

GENERAL PARALYSIS IN THE SENILE PERIOD, WITH A
 (226) **REPORT OF TWO CASES, INCLUDING POST-MORTEM**
EXAMINATION. M. J. KARPAS (of New York), *N.Y. Med.*
Journ. Jan., 25, 1908.

THE report of the clinical histories and anatomical findings in the case of two female paralytics, aged sixty-five and seventy respectively.

The similarity of the picture to that of senile dementia is noted, and the importance of the examination of the cerebro-spinal fluid emphasized. The duration of the disease in the first case was five years. In both cases there was definite megalomania.

C. MACFIE CAMPBELL.

ALCOHOLISM AND INSANITY. (*Alcoholisme et Folie.*) M.
 (227) LEGRAIN, *Presse Méd.*, Jan. 29, 1908.

IN this paper a somewhat detailed account is given of the results of a statistical inquiry carried out by Amaldi in Italian asylums regarding the relations of alcoholism and insanity, and these are compared with statistics obtained in France. Numerous tables are given.

A. HILL BUCHAN.

ON PAINS IN MANIC-DEPRESSIVE INSANITY. (*Ueber Schmerzen*
 (228) *beim manisch-depressiven Irresein.*) J. SCHROEDER (of Roth-
 enberg), *Centralbl. f. Nervenhe. u. Psych.*, Dec. 15, 1907.

THE author calls attention to the occurrence in manic-depressive patients of attacks of pain, fairly definitely localised and of much longer duration than attacks of migraine. These pains usually occur during the period of recovery, frequently after discharge. They do not show the life-long periodicity of attacks of migraine, nor are they so severe. Schroeder reports a case which illustrates the occasional difficulty of diagnosis due to such pains.

C. MACFIE CAMPBELL.

THE MENTAL DISORDERS OF ANCHYLOSTOMIASIS. (*As*
 (229) *Desordens Mentais na Ankylostomíase.*) A. ANSTREGÉSILO
 and H. GORUZZO, *Arch. Brasil. de Psychiatr., Neur. e Sciencias*
Affins., 1907, Nos. 3 and 4.

MANY cases have been recorded in which irritation of the intestines by worms has been assigned as the exciting cause of reflex psychoses, but, notwithstanding this popular belief in the connexion between intestinal worms and acute maniacal states, Krafft-Ebing

and others have come to the conclusion that insanity due to worms is an exceedingly infrequent manifestation, and when it does occur is mostly in children of neuropathic constitution. With this conclusion the authors of this short but interesting paper are in agreement. They describe three cases in which the patients suffered from grave psychoses, apparently due to anchylostomiasis, all of whom made good recoveries after anthelmintic treatment by thymol. The authors leave out of count many cases they have observed of mild alteration in the mental character associated with the same parasitic condition. In all cases the characteristic anæmia was present, blood counts showed great deviations from the normal, and the ova of the *uncinaria duodenalis* were present in the fæces. All cases were stated to be of degenerate type. As to type of mental disorder: the first case was depressed and had delusions of persecution; the second case showed pronounced alterations in memory, with verbal incoherence and religious delusions; and the third case, marked mental confusion with almost complete disorientation. In none of the cases was there a history of syphilis, alcoholism or other toxic agents apart from those generated in the intestines or tissues of the body, presumably as the result of the anchylostomiasis or the consequent intestinal catarrh. The authors conclude that anchylostomiasis should be considered as a cause of mental disorder, these being of two kinds, the first frequent and mild, consisting mainly in slight alteration in the character; and the second much less frequent but of a grave kind, their appearance being determined by an original degenerescence and the existence of the anchylostomum.

R. CUNYNGHAM BROWN.

PSYCHASTHENIA : ITS SEMEIOLOGY AND NOSOLOGIC STATUS
(230) **AMONG MENTAL DISORDERS.** J. W. COURTNEY (of Boston),
Journ. of Amer. Med. Ass., Feb. 29th, 1908.

In introducing the subject the author refers to the vagueness of the term psychasthenia and the struggle it has had and is still having to gain recognition as a nosological entity. The phenomena by which it is distinguished are: Obsessions—of sacrilege, of crime, of disease, of shame of self, of shame of body; compulsions—of ideation and activity; manias of going to the extreme—of precision, of verification, of orderliness, of symmetry, of contrast and contradiction, of cleanliness, micromania, arithmetical mania, mania of symbols, of research, of explanation, etc.; enforced reverie; crises of agitation; phobias; anxious states. All of these phenomena do not appear in every case, but whether they appear singly or in groups they belong to one clinical picture and should have one clinical designation.

In its pathology one must expect and will find morbid ideation and morbid activity of a wide range. About 75 per cent. of cases come from families in which some form of mental alienation has appeared in more than one generation. The early environment and surroundings have much to do in directing the growth of the personality into morbid channels.

Psychasthenia is differentiated from hysteria by the fact that "the disturbance of consciousness which marks it is general"; from neurasthenia "by the patient being too much absorbed by obsessions, tics, etc., to dwell upon fatigue, paræsthesias, insomnias, which mark the presence of this disease." Finally, he reaches the conclusion that "psychasthenia is a *forme fruste* of intellectual petit mal."

The paper is long, deals much with generalisations, and furnishes little that is definitely new. C. H. HOLMES.

**OBSERVATIONS ON THE OPSONIC INDEX TO VARIOUS
(231) ORGANISMS IN CONTROL AND INSANE CASES. C. J.
SHAW, *Journ. of Ment. Sc.*, Jan. 1908.**

So far as is at present known, the opsonic index of the blood serum of healthy persons to the majority of organisms is similar to the tuberculo-opsonic index. In this paper the average opsonic indices for five consecutive days of healthy non-tuberculous control cases to the tubercle bacillus, bacillus coli communis, staphylococcus aureus, and micrococcus rheumaticus are compared with those of insane patients in good bodily health, and the observations were continued after the injection of Koch's new tuberculin T. R.

There was found to be little difference between the average indices of the control cases to the various organisms, but in the insane the indices obtained to bacillus coli and staphylococcus aureus were rather higher than those to the tubercle bacillus and micrococcus rheumaticus, but, except in the case of bacillus coli, the control index was higher than that of the insane to each organism. After the injection of 1/500 mgr. T. R., a negative phase was found to occur to the other organisms as well as to the tubercle bacillus in both the control and insane cases, but, except to staphylococcus aureus, the percentage of negative phases was less in the control cases than in the insane. One control case was injected with 1/750 mgr. T. R., the only negative phase produced being to micrococcus rheumaticus. In three insane patients similarly injected negative phases resulted to all the organisms, though in a less percentage than when the larger dose was administered. On comparing the results obtained in the control cases and in the acutely and chronically insane patients

before injection, the average indices are found to be lowest in the acutely insane to all the organisms, while the average of the chronic cases is below that of the controls to the tubercle bacillus and micrococcus rheumaticus, but slightly above the control index to bacillus coli and staphylococcus aureus. The aggregate average of the control cases, however, was 1.06, while that of the chronic cases was 1.01. The proportion of negative phases produced by the injection of T. R. was least in the control and greatest in the acutely insane cases. It is therefore concluded that the resistant power of the insane to organismal invasion is less than that possessed by ordinary individuals, and that the acutely insane are more liable to infection than the more chronic cases.

AUTHOR'S ABSTRACT.

TREATMENT.

OPERATIVE PROCEDURE AS A THERAPEUTIC MEASURE IN
(232) **EPILEPSY.** M. Woods (of Philadelphia), *Journ. of Amer. Med. Ass.*, Feb. 29th, 1908.

EPILEPSY is defined as a paroxysmal apyretic neurosis in which its victims are easily impressed neurotics. Any sudden shock creates a violent impression on their nervous system, and recoveries following operations result more from the shock than from the tangible thing that has been removed or corrected. Any surgical procedure whatever may be followed by improvement or recovery from the epileptic attacks.

The author reports three cases of his own in which respectively an amputation of the arm, a severe wound of the thigh, and an operation for tuberculosis of the glands were followed by apparent recovery from these attacks. Dr J. Wm. White records ninety cases in which trephining was performed, no lesion found, and nothing particular done, yet marked relief and complete cure followed.

The author finally concludes that much benefit would be derived by the patient if in all such cases where operation is considered, the neurologist would consult with the surgeon.

C. H. HOLMES.

SEVERE SPASMODIC CONTRACTION OF A FINGER CURED BY
(233) **STRETCHING THE MEDIAN NERVE.** JAMES ADAM, *Lancet*,
Feb. 1, 1908, p. 287.

THIS patient, a woman of forty-five, had her left hand amputated for a septic infection. Two and a half years later the middle finger of the right hand began to contract; there were no signs of disease

in the finger; with some force and difficulty it could be extended, but it gave great pain, and when released the finger went back like a spring with the tip firmly fixed in the palm. This finger was amputated. In six months a similar condition developed in the ring finger. There was the same strong spasmodic contraction, which could be with great pain and force overcome, but there was no power of voluntary movement. It was not red, swollen, or painful, and its sensibility was normal, and the other fingers were normal in every way. Stretching of the median nerve was suggested; this was done in the upper arm, both distal and proximal parts being forcibly stretched for four or five minutes. For nearly a month the patient had numbness and disordered sensation in, curiously, the little and ring fingers, but none in the thumb or forefinger. Splints and passive movement were employed for a little. When seen a year later the finger could be bent and extended normally, and there was no tendency to contraction, so that the cure may be regarded as permanent. The etiology of the case is unexplained.

J. H. HARVEY PIRIE.

THE PATHOGENESIS AND TREATMENT OF NEURASTHENIA

(234) **IN THE YOUNG.** R. N. WILLSON, *Am. Journ. of Med. Sc.*, Feb. 1908, p. 178.

THE author casts overboard "nerve-stress and nerve-tire" as causes of neurasthenia. They are merely symptoms; the primary process is to be looked for in a general lowering of tissue nourishment, and the upbuilding of this is *the* means of cure. In all cases of neurasthenia in the young the beginning is due to some definite cause which, if removed in time, will admit of a cure of the patient. Four cases are narrated with, rather scrappily, the means adopted for treatment—active hygiene with graduated exercise in the open air to the point of physical tiredness being apparently the main feature.

J. H. HARVEY PIRIE.

Reviews

OUTLINES OF PSYCHIATRY. WM. A. WHITE (of Washington), New York: Nervous and Mental Disease Monograph Series, No. 1, 1908, pp. 232, price \$2.

THE purpose of the book is to give in a condensed and simplified form the essentials of psychiatry which would be useful to the medical student and the young practitioner in acquiring a working knowledge of the subject. It is a neatly printed volume of two

hundred and thirty-two pages, divided into eighteen chapters ; the first seven are devoted respectively to a brief psychological introduction, definition of insanity, classification, its causes, treatment, symptomatology, and a scheme for examination ; the last eleven chapters take up the individual forms of insanity and discuss each briefly but concisely.

The chapter on the examination of the insane is one of the most valuable which the book contains ; it is direct, comprehensive, and should be of service not only to the student but to those more advanced in the subject.

The classification deals only with the principal groups recognised in the newer psychiatry ; vague and ill-defined terms are omitted. In treatment hydrotherapy is given a deservedly prominent place.

Altogether the " Outline " is a clear, systematic, conservative, well-edited work, which should help to meet the popular demand for a brief outline in English of the newer psychiatry.

C. H. HOLMES.

ÉTUDE MÉDICO-PSYCHOLOGIQUE SUR THOMAS DE QUINCEY.

PAUL GUERRIER, Lyon, Rey, 1907.

IN this interesting study the author has analysed the life and work of Thomas De Quincey from the point of view of the physician, and comes to some interesting conclusions. The family from which De Quincey sprang was full of psychopathic personalities. Almost all the members showed unstable nervous equilibrium, and the disorder of the nervous system present in his brothers and sisters was still more pronounced in the person of the essayist himself. In early boyhood there was evident a morbid sensibility which left marked traces in his later writings. He was essentially a " visual," and in the dreams of the Confessions it is what he has seen, and not what he has heard, that he describes so vividly. The details of memories of the earliest age were deeply engraven on his mind. His health as a boy was indifferent, and at the age of sixteen symptoms of gastric intolerance developed, which never left him. To judge from the symptoms the disorder was of a nervous nature. At a later period of his life he tended to give a hypochondriacal misinterpretation to these symptoms. At the age of twenty-two he had profuse sweats and attacks of dyspnœa. After a deeply emotional incident at the age of twenty-seven he had hallucinations, and developed a transitory neurosis characterised by intense anxiety. The disorder disappeared abruptly. For months after being bitten by a dog he was unable to work owing to the obsession of hydrophobia. From what De Quincey himself has written, one would

imagine that he was a confirmed opium eater who took the drug in quantities never equalled. As a matter of fact he does not seem to have suffered from the usual clinical symptoms of chronic opium poisoning, and what we know of his life and long intellectual activity is absolutely incompatible with his statements as to the amount of opium consumed. His writing never showed any tremor. He was able at the age of seventy to walk uphill with great rapidity. He showed no evidence of premature senility. His memory remained always extraordinary. During the periods when he claimed to have given up opium, there is no trace in his correspondence of the well-known symptoms of abstinence in the chronic opium eater. The author concludes that De Quincey was a psychopathic individual of morbid sensibility with a general hyperæsthesia, with a love of the mysterious and a delight in the products of the imagination, showing traces in his work of that mythomania which is so frequent among the hysterical. To a certain extent the opium habit was a myth, but it pleased his imagination and gave him a literary pose which was not without a market value.

The book is delightfully written, is not interrupted by too frequent references to actual documents, presents the view of the author in a simple and lucid manner. It is somewhat lighter than similar studies by Germans on various psychopathic men of genius. It is an interesting appreciation of the various factors which went into the composition of the works of the great essayist, although the author's neglect of many available sources of information makes one cautious in accepting without reserve his conclusions.

C. MACFIE CAMPBELL.

BOOKS AND PAMPHLETS RECEIVED.

Riv. de Medicina y Cirugia practicas. Feb. 1908. Cardona, Madrid.
Siegfried Weinberg. "Über den Einfluss der Geschlechtsfunktionen auf die weibliche Kriminalität." Marhold, Halle, 1908, M. 1.

W. v. Bechterew. "Die Funktionen der Nervencentra." *Deutsche Ausgabe.* Erstes Heft. Fischer, Jena, 1908, M. 16.

P. Sainton et L. Delherm. "Les Traitements du Goitre Exophtalmique." Baillière et fils, Paris, 1908, 1 fr. 50 c.

Hans Gudden. "Ueber Massensuggestion und psychische Massenepidemie." Verlag der Aertzlichen Rundschau. München, 1908, M. 0.75.

Harris E. Santee. "Anatomy of the Brain and Spinal Cord with Special Reference to Mechanism and Function." Fourth Edition, revised and enlarged. Sidney Appleton, London, 1908, 16s.

Archibald Church. "Modern Clinical Medicine. Diseases of the Nervous System." Authorized Translation from "Die deutsche Klinik," Ed. Julius L. Salinger. Sidney Appleton, London, 1908, 28s.

Review of Neurology and Psychiatry

Original Articles

A CASE OF ACUTE ASCENDING PARALYSIS OF SYPHILITIC ORIGIN.

By O. CROUZON, Chef de Clinique de la Faculté à l'Hôtel Dieu de Paris,
and

GEORGES VILLARET, Ancien Externe de l'Hôpitaux de Paris.

THE following case of acute ascending paralysis of syphilitic origin appears to us worthy of publication. The interest of such a case lies first of all in the rarity of this syphilitic affection of the nervous system, and in this particular case there are especial points of interest in the history to which we shall later direct attention.

The patient was 42 years of age. He had contracted syphilis at the age of 32. We saw him on one occasion, April 1906, with Dr P. Wiart, for an ulceration of the frænum of the prepuce, which presented certain features which led one *à priori* to consider it a syphilitic chancre. The lesion was, as a matter of fact, slightly indurated and ulcerated, the basis of the ulcer presenting a beefy red appearance. There was, however, no affection of the inguinal glands, and we made the diagnosis of chancriform gumma of the frænum of the prepuce. The treatment which was instituted confirmed the diagnosis. After a series of ten intra-muscular injections of biniodide of mercury the ulcer healed.

R. OF N. & P. VOL. VI. NO. 5—X

In October 1907 the patient, whom we had advised to follow from time to time mercurial treatment, consulted us again and was given a series of ten intra-muscular injections of one cubic centimetre of biniodide of mercury (a watery solution containing one centigramme in each cubic centimetre). The patient talked to us incidentally of vague sciatic pains which he had been feeling for several months in the right inferior extremity.

These pains persisted and became more severe, so that in December 1907 we examined him from this point of view, and found marked pain on pressure over the characteristic points, the sciatic notch, the trochanteric groove, and behind the external malleolus ; raising the extremity, with the knee-joint extended, caused acute pain (sign of Lasègue).

Different forms of treatment were followed in order to cure this sciatica, but none gave any result. As a last resort we prescribed, early in January 1908, Dupuytren's pills to be taken two daily for twenty days. The sciatic pains, however, became daily more severe, and finally produced a true functional paralysis of the right inferior extremity. In view of the failure of the treatment, we decided on January 31, 1908, to again adopt more energetic measures. We were on the point of beginning a new series of intra-muscular injections of large doses of biniodide when, on February 4, 1908, the patient sent for us on account of symptoms which he ascribed to influenza, and at first they seemed to us to depend on that disorder. He complained of general fatigue, pains in the limbs, and headache ; he was feverish ; one symptom dominated all the others, namely, torticollis of the muscles of the neck, so that it was impossible for him to move his head in relation to the trunk. Pressure of the muscular mass of the back of the neck caused very acute pain ; on the other hand, pressure on the vertebral column, and in particular on the cervical portion, did not cause the slightest pain.

The same symptoms continued without modification during February 6th and 7th.

February 7th.—We note the appearance of a new symptom, which consists in a paresis of the superior and inferior limbs on the right side. This paresis is more marked in the lower than in the upper limb. It appears in the upper limb to affect particularly the extensor group of muscles, while the movements of opposition of the thumb, the movements of flexion of the

fingers on the hand, of the hand on the forearm and of the forearm on the arm, appear satisfactory. The weakness, therefore, is a mild degree of paralysis of radicular origin. The face, on the other hand, is completely spared. There is no impairment of movement of the muscles of the eyes, of the mouth, and of the tongue. The tendon reflexes are normal.

February 8th.—The same symptoms persist and are more marked. To-day we have to do with a well-marked paralysis of the superior and inferior limbs of the right side. In addition there are the following symptoms—incontinence of fæces, retention of urine, rise of temperature ($40^{\circ}3$ C. in the rectum).

In view of the serious and progressive nature of these symptoms we diagnose *acute ascending paralysis*, and, while still in doubt as to the nature of the affection, we give an injection of 3 centigrammes of biniodide of mercury, which is continued daily. From this day onwards the patient requires to be catheterized twice daily.

February 9th.—The temperature remains raised, is $40^{\circ}2$ in the rectum; the pulse is 96, the respirations 35 per minute. The paralysis of the superior and inferior limbs becomes more marked. The patellar reflex is absent on both sides. Examination of the plantar reflex discloses a well-marked sign of Babinski on the right side, and the same sign still more marked on the left side. Notwithstanding this there is not at present, and has never been, any paralysis on the left side. Sensibility is, and has always been, intact in all its forms.

Since he has been confined to bed the patient has assumed the dorsal decubitus, the only position which allows him to keep his head at rest. From this day onwards it appears that the movements of the head from right to left and in the opposite direction are less painful, if care be taken that the head is lying on a pillow.

Endeavours to flex the thighs on the pelvis, while the legs are extended on the thighs, cause such extreme pain that these movements of flexion cannot be carried further.

February 10th, morning.—The rectal temperature is $40^{\circ}3$. The movements of the forearm on the arm, of the hand on the forearm, and of the fingers on the hand are slowly reappearing. It is the same with the patellar reflex, which is now elicited on

the left side ; it is still absent on the right side. The cremasteric reflexes are still absent.

3.30 P.M.—Lumbar puncture is performed. A clear crystalline fluid issues ; the cytological examination of the fluid, which is made one hour later, demonstrates a definite spinal lymphocytosis ; only an occasional polymorphonuclear is to be found in the centrifuge deposit.

7 P.M.—The rectal temperature is $39^{\circ}3$.

February 11th, morning.—The rectal temperature is $38^{\circ}3$, the pulse is 84. We note a very considerable amelioration of the symptoms so far as the extensors of the right superior extremity are concerned ; the extension of the fingers is of wider range. The condition of the tendon reflexes and of the plantar reflex remains the same.

Evening.—There is progressive improvement. Since noon the patient has been able, for the first time, to retain his fæces ; he is still unable to urinate, and we are still forced to catheterize him. From every other point of view there has been no change.

February 12th, morning.—The temperature in the axilla is $38^{\circ}3$, the pulse is 84.

The superior extremity has almost completely recovered the range of the movements of extension and of flexion of its different segments. The right inferior extremity still remains inert on the bed. On the left side the patellar reflex is definitely elicited ; on the right side there is occasionally the suggestion of a reflex ; the cremasteric reflexes are still absent on both sides. Unfortunately during the night incontinence of fæces again appeared.

Evening.—The axillary temperature is $39^{\circ}3$, the pulse is 92. The condition is exactly similar to that in the morning.

February 13th, morning.—The axillary temperature is $39^{\circ}3$, the pulse is 92.

None of the symptoms mentioned above have varied ; nevertheless there appears to be slight improvement, since we notice a faint movement of abduction of the right thigh on the pelvis, whereas this thigh had previously remained quite powerless.

4.30 P.M.—The axillary temperature is always $39^{\circ}3$, the pulse is 92 ; respirations, 40 per minute. We observe that there is some mental clouding which is quite transitory. There has never been any vomiting.

Evening.—We observe no change in his general condition ;

the axillary temperature is $37^{\circ}6$, the respirations are still 40, and the pulse 84 per minute.

February 14th, morning.—The axillary temperature is $39^{\circ}2$, the pulse is 88, and the respirations 32 per minute. We again notice some mental clouding, which is more pronounced and less transitory; in addition, there is a certain degree of slurring in the speech.

On the same day, at 7 P.M., we find the patient in a state of coma, with tracheal râles, and lying "*en chien de fusil*." The pupils no longer react to light. From 7.15 P.M. onwards the following is the course of temperature, pulse, and respiration:—

7.15 P.M.	{	Temp.: in axilla, $41^{\circ}6$.
		Pulse: 108 per min.
		Resp.: 54 per min.
7.40 P.M.	{	Temp.: in axilla, $41^{\circ}7$.
		" in rectum, $41^{\circ}7$.
		Pulse: 112 per min.
		Resp.: 52 per min.
7.55 P.M.		Resp.: 54 per min.
8.40 P.M.	{	Temp.: in axilla, $42^{\circ}1$.
		" in rectum, $42^{\circ}1$.
		Pulse: 110 per min.
		Resp.: 48 per min.

All the cutaneous and tendinous reflexes are absent.

9.15 P.M.		Temp.: in rectum, $42^{\circ}1$.
9.40 P.M.	{	Temp.: in rectum, $42^{\circ}2$.
		" in axilla, $42^{\circ}2$.
		Pulse: 116 per min.
		Resp.: 44 per min.

Death at 10.55 P.M.

11.5 P.M., 10 minutes after death—

Temp.: in axilla, 43° .
 „ in rectum, 43° .

To sum up, we have had to do with a patient who, after having suffered for several months with sciatica on the right side, was suddenly affected with paresis of the superior and

inferior extremities on the right side, the onset being accompanied by the symptoms of an acute infection. These symptoms were progressive for two days, and were accompanied by incontinence of *fæces*, retention of urine, disorders of the reflexes, and elevation of temperature. Then, for the following two days, there was a slight tendency to improvement, and finally, during the last three days of life, the original symptoms became more marked, and we observed a slight mental clouding. At the same time the temperature rose, as is usual in the affections of the nervous system, where the thermic centres of the medulla oblongata appear to be affected.

The disease, therefore, is one which ran in eight days a course similar to that of the acute ascending paralyses.

Two questions must at the start be definitely formulated. What was the nature of this acute ascending paralysis? On what anatomical localisation did it depend?

The nature of this ascending paralysis is made clear to us through the lymphocytosis of the cerebro-spinal fluid; it allows us, as a matter of fact, to exclude all other infections except tuberculosis and syphilis; now, in the history of the disease we find nothing to suggest the course of tuberculous meningitis. On the other hand we have exact knowledge as to the syphilitic history of the patient, admitted by himself and revealed by the chancriform gumma of the *frænum*.

The anatomical localisation of this acute ascending paralysis can be deduced on the one hand from the state of the cerebro-spinal fluid, and on the other from the clinical picture; the lymphocytosis allows us, as a matter of fact, to affirm that there has been meningitis; the clinical picture has shown us the existence of a paralysis of radicular type in the superior extremity; there has therefore been present not merely meningeal inflammation, but also nerve-root inflammation. Finally, the affection of the sphincters, the rise of temperature, and the terminal mental disorders allow us to affirm that the centres themselves have been affected.

We believe, then, that we are justified in concluding that it is a case of meningo-radiculo-myelitis of syphilitic origin which followed a sciatica, itself perhaps of syphilitic nature, and that the clinical picture has been that of an acute ascending paralysis, ending fatally after a course of eight days.

We have examined medical literature to see whether there have been reported any cases comparable to ours. With this aim in view we have consulted several authors, Williamson,¹ Rosin,² the two latest monographs on syphilis of the nervous system, that of Lamy³ and that of Nonne.⁴

Nonne says that there are cases of Landry's paralysis of syphilitic origin, following the type described by Goebel and Von Hartog, with paralysis of the inferior extremities, bulbar paralysis, disorders of sensibility, and loss of the reflexes. Landry himself, in one of his ten cases, found syphilis. Kussmaul has described several cases of Landry's paralysis with a history of syphilis in the parents. Alexandre, in his book "Syphilis et Yeux," describes an analogous case.

Heubner, on the basis of his own observations, and those of Zambaco, Léon Gros, and Lancereaux, describes a form of Landry's paralysis which appears without prodromata and without meningitic phenomena at an early stage of syphilis.

Our case is comparable to the above in its clinical appearance, but in it we had the good fortune to observe active syphilitic manifestations one year before the ascending paralysis, and at the very moment when there was developing a sciatica, which itself was perhaps syphilitic.

On the other hand, we have been able to add to our clinical record one observation which can not be found in the old records—namely, the result of lumbar puncture; the lymphocytosis of the cerebro-spinal fluid confirmed our clinical diagnosis of syphilitic meningitis, and in consequence gave much greater precision to the etiology of our case.

We wish finally to direct attention to one interesting feature of the temperature curve; the temperature, which before death reached 42°·2, as happens in many fatal affections of the nervous system, rose still further after death and reached 43°; this fact is also well known. But the most interesting point appears to us to lie in the fact that, at the terminal period of the disease the temperature, examined on four different occasions, reached the same height in the axilla as in the rectum:—

¹ Williamson, *Edinburgh Medical Journal*, October 1900.

² Rosin, *Zeitschrift für klinische Medizin*, 1896.

³ Lamy, "Syphilis des Centres Nerveux." Collection Léauté.

⁴ Max Nonne, "Syphilis und Nervensystem," Berlin, 1902.

7.40 P.M.	Temperature in axilla,	41°·7.
	„ in rectum,	41°·7.
8.40 P.M.	„ in axilla,	42°·1.
	„ in rectum,	42°·1.
9.40 P.M.	„ in axilla,	42°·2.
	„ in rectum,	42°·2.
11.5 P.M.	„ in axilla,	43°.
	„ in rectum,	43°.

The two temperatures have been identical on each occasion.

There is thus present a disturbance in the central and peripheral temperature which appears worth attracting attention. We have looked in the work of Bourneville¹ to see whether there exist cases of this kind in the terminal period of cerebral hæmorrhage, of cerebral softening, of eclampsia, and of epilepsy, and we have only found temperatures extremely high, but no mention is made of cases with identical temperature in the axilla and in the rectum.

Eichhorst,² however, has stated that in certain very rare cases, as to which, however, he gives no details, the axillary temperature may exceed by one degree the temperature in the rectum.

The phenomenon in such a case is perhaps due to the absence of regulation, owing to involvement of the thermic centres in the medulla, and the symptom should then be considered as a bulbar trouble which has characterised the terminal period of the ascending paralysis.

But apart from this thermometric peculiarity the clinical observation has appeared to us to be specially interesting on account of the syphilitic origin of this ascending paralysis.

¹ "Étude clinique et thermométrique sur les maladies du système nerveux," Paris, 1873.

² Eichhorst, "Traité de Diagnostic Médical."

Abstracts

ANATOMY.

THE STRUCTURE OF GREY MATTER. JOHN TURNER, *Brain*, (235) 1907.

THIS paper, the last of a series on a similar topic which have appeared in *Brain* and elsewhere, seeks to establish in grey matter, not only two distinct kinds of nerve cells—the “pale” or ganglionic, and the “dark” or intercalary, but proceeding from them respectively two kinds of neurofibrils, one of smooth contour, the other with beads along its traject.

An endeavour is also made to establish the network character of the periganglionic beaded fibrillar investment, which is shown to be derived from the ultimate branchings of the intercalary cells. As this structure is manifestly recruited on all sides by fibrils from different intercalary cells, it follows, if this can be proved, that neurofibrils are continuous, and therefore at some places beaded neurofibrils should pass over into smooth contoured neurofibrils, as is shown to be the case.

So far as possible, the appearances shown by the writer's methylene blue and peroxide of hydrogen method were collated with those obtained from tissues treated by Cajal's reduced silver method, but although this latter occasionally selects the intercalary cells, it only imperfectly and rarely does so, and still more rarely does it select the beaded neurofibrils.

The scheme of the nervous system on the lines of continuity of fibrils, suggested by the writer a year ago in *Brain*, is further amplified, and he believes that it is quite capable, by means of the periganglionic networks, of meeting the requirements of the known laws of nervous conduction.

AUTHOR'S ABSTRACT.

PATHOLOGY.

ON REGENERATION IN THE PERIPHERAL SEGMENT OF A
(236) **NERVE PERMANENTLY SEPARATED FROM ITS CENTRE.**

(Zur Frage der Regeneration in einem dauernd von seinem Zentrum abgetrennten peripherischen Nervenstumpf.) A. MARGULIES (of Prague), *Virch. Arch.*, Bd. 191, Hft. 1, January 1908.

THE author comes to the following conclusions:—1. After section of a peripheral nerve definite degenerative changes occur in the distal segment; the axis-cylinder and medullary sheath completely

disappear. 2. The cells of Schwann increase in size and number, and form a new specific fibrous tissue. 3. The nerve remains in this incomplete condition, if separation from the centre is permanent. 4. If its connection with the centre is restored, it becomes differentiated into a structurally complete nerve with axis-cylinder and medullary sheath. 5. Autogenous regeneration, *i.e.* the formation of complete nerves, does not occur in the distal segment in the grown animal if the division of the nerve is permanent. 6. Every regeneration of nerve is an autonomous process of growth in so far as the anatomical basis of the nerve is furnished by the cells of Schwann. HART (C.g.B.).

CLINICAL NEUROLOGY.

DIAGNOSIS OF ORGANIC FROM FUNCTIONAL AFFECTIONS
 (237) **OF THE NERVOUS SYSTEM.** J. S. RISIEN RUSSELL, *Brit. Med. Journ.*, March 14, 1908.

CASES where the question arises of differential diagnosis between organic disease and functional disturbance of the nervous system fall into one of four classes: First, cases of organic disease showing physical signs characteristic of organic disease; second, functional cases with physical signs indicating the functional nature of the affection; third, cases of organic disease with no physical signs characteristic of such; and fourth, cases of functional disorder without physical signs typical thereof. Cases falling into the first two classes are comparatively simple. The second two classes present greater difficulties.

1. *Organic disease, with physical signs.*—Ophthalmoscopic examination may reveal the presence of optic neuritis or atrophy. These are never found in uncomplicated functional conditions. Absence of the knee-jerks or presence of the extensor type of plantar reflex justifies a diagnosis of organic trouble. Presence of the extensor plantar reflex is often the chief sign diagnostic of disseminated sclerosis as opposed to hysteria or neurasthenia.

Care must be taken not to misinterpret physical signs.

Exaggeration of the knee-jerks must not be regarded as necessarily due to an organic lesion, unless associated with a true ankle clonus, the extensor plantar reflex, or other undoubted sign of organic disease. Any associated abnormality must also be correctly interpreted. For example, exaggeration of the knee-jerks associated with pes cavus does not of necessity mean spastic paraplegia, nor if nystagmus be present with it, is a diagnosis of disseminated sclerosis necessarily correct. Signs of organic disease may appear late in the history of the case. It often happens that

pains in the lower limb, which at first appear to be due to sciatica, ultimately prove to be due to organic pelvic trouble. It should be remembered that when there is only functional disturbance of the sciatic nerve, the ankle-jerk is commonly increased; whereas, when the nerve is damaged, the jerk is diminished or abolished.

Cases regarded as neurasthenia in the early stages may, after many months, show signs of organic nerve disease.

2. *Functional affections, with physical signs.*—In cases of hysterical paralysis, the muscles antagonistic to the paralysed muscles may often be found to be in action, when the attempt is made to use the paralysed muscles. Thus, supposing the quadriceps extensor to be affected, if, with the patient in bed, the observer flex the limb, resist the patient's attempt to extend it, and then withdraw the resistance, in the case of organic paraplegia the limb falls to the bed in the extended position; whereas, in the case of hysterical paralysis, the limb may remain slightly flexed because the hamstring muscles are in action.

In other cases, the knee and ankle jerks may remain normal where one would expect a change were the condition organic, or anæsthesia may be present where the possible organic disease suggested would show none.

3. *Organic disease, without physical signs.*—The best example in this class is paralysis agitans in its early stages. In a case which looks like one of neurasthenia, loss of power confined to one side or to one limb, stiffness of the limbs or inequality of the tendon jerks on the two sides should suggest the possibility of paralysis agitans. The knee-jerk is often more active on the side on which the paralysis agitans begins, and the ankle-jerk diminished or absent on the same side. Diagnosis of paralysis agitans may be quite justified although the patient be young, and though the tremors be in abeyance when the patient thinks herself unobserved. Tremors may sometimes be noticed in the muscles of the face, tongue, or jaw of a nature characteristic of paralysis agitans. There is often an indefinite something in the general appearance and mode of progression strongly suggestive of paralysis agitans.

Diagnosis in this class of case is often more difficult because the circumstances of onset may be those under which functional affections commonly arise.

4. *Functional affections, without physical signs.*—Under this head a case is cited where chorea, a diagnosis of which seemed otherwise to be the natural conclusion, was excluded by the fact that the speech affection which was present was not at all like that characteristic of chorea. Another case is mentioned where the presence of organic disease was negatived by the complete absence of any physical sign characteristic of organic disease, by

the fact that station and locomotion, while abnormal, were not typical of any particular organic disease.

In these cases, the faradic current and the assurance that it would be effective, cured the condition. J. M. DARLING.

EARLY OSTEO-ARTHRITIC MANIFESTATIONS OF TABES. (Le (238) *Tabes Ostéo-articulaire Précocce, etc.*) STEFANI, *Gaz. des Hôp.*, Feb. 25, 1908, p. 267.

THIS article deals with bone and joint affections in the pre-ataxic stage of tabes. The author recognises three forms: the first, where the manifestations are purely osseous, principally in the form of spontaneous fractures, but also as localised osteitis, especially in the small bones of the extremities; the second, where the lesions are purely in the joints—tabetic arthropathy; and the third, which is a combination of the two, before the appearance of any of the classical signs of tabes. He gives a full description of a case of the last type, a woman of 54, who within two years had three spontaneous fractures of the lower extremity, and associated bony changes in the foot and knee-joint. At the time of the last fracture she showed also some inequality of the pupils, sluggish reaction to light, and pains of the lightning character; these were the sole tabetic symptoms. Fractures in these cases heal but slowly, and with great excess of callus, the callus being remarkably transparent to X-rays.

J. H. HARVEY PIRIE.

SCOLIOSIS IN INFANTILE PARALYSIS. (Skoliose bei Kinder- (239) *lähmung.*) P. EWALD, *Zeitschr. f. Orthopäd. Chir.*, Bd 19, Heft 3 u. 4, S. 549.

THE author has had an opportunity of investigating the rare condition of pure paralytic scoliosis. The case was that of a child of $2\frac{4}{12}$, which had a feverish affection during the first year of life, which left the right leg completely paralysed; the child never stood or walked, scarcely ever even sat up, but lay constantly in the horizontal position. Nevertheless, a complete scoliosis with the convexity to the left developed, with bulging of the ribs and lumbar region on the left. The convexity extended from the 1st dorsal spine to the sacrum, the 11th dorsal spine being furthest from the middle line—2.5 cm. There was marked rotation of the bodies of the upper lumbar and lower dorsal vertebræ, so that their left transverse processes lay in the same frontal plane as the tips of their dorsal spines. The curvature of the spine as seen from the front was therefore much greater than the displacement of the dorsal spines would appear to show.

The affected muscles of the back were all of the left side, viz., the multifidus and semispinalis dorsi, the rotatores, interspinales, and intertransversales. He shows fairly conclusively that the resulting deformity is the result of the continuous unopposed action of the transverso-spinales and small, deep muscles of the right side.

J. H. HARVEY PIRIE.

TUMOURS OF THE CAUDA EQUINA AND LOWER VERTEBRÆ.

(240) WILLIAM G. SPILLER, *Amer. Journ. Med. Sc.*, March 1908.

THE author discusses the differential diagnosis between hysteria, multiple neuritis confined to the lower limbs, intra-pelvic tumour, tumour or caries of the lumbar vertebræ or sacrum, lesions within the vertebral canal but external to the dura, tumour or other lesion (hæmorrhage) of the conus, and tumour of the cauda equina. The paper contains the report of nine cases; seven with necropsy, three with operation.

In case 1 a round-cell sarcoma was found within the roots of the cauda equina. Case 2 was especially interesting, as the tumour (fibro-sarcoma) was at the centre of the cauda equina, and yet the nerves for the bladder and rectum escaped, and the pain at first was unilateral. The symptoms made the clinical diagnosis between tumour of the cauda equina and tumour of the vertebræ difficult. In case 3 numerous hard masses (osteo-sarcoma) were found in the roots of the cauda equina, and most of these were in the roots where they penetrated the dura, although some were within the roots in the dural cavity. A fibro-sarcoma was found in case 4 outside the dura, and an endothelioma in case 5, also external to the dura. Cases 6 and 7 were clinical and without confirmation by operation or necropsy. In addition to these seven cases brief reference is made to a lipoma of the filum terminale and to a small osteoma of one of the roots of the cauda equina.

Statistics as yet do not justify the statement that the prognosis from surgical intervention on the sacrum and lumbar vertebræ is decidedly better than on the other vertebræ and the cranium. In the author's cases, when the tumours could be examined they were all of such a character that complete removal would have been impossible, and this seems to have been true of most, if not of all, recorded cases. The well-defined almond-shaped fibromas or fibro-sarcomas occurring frequently at higher levels of the cord are much less likely to develop in the region of the cauda equina. If the prognosis for surgical treatment at present is somewhat gloomy, a larger experience may give reason for hope. It is possible at least to cut posterior roots in order to relieve pain. A large amount of cerebro-spinal fluid escapes when the lower

part of the spinal column is opened, and urine and fæces are likely to soil the bandages in those cases where the bladder and rectum are paralysed, although it should be possible to prevent this. The paper is a long one, and does not lend itself readily to abstracting.

AUTHOR'S ABSTRACT.

EPIDEMIC CEREBRO-SPINAL MENINGITIS IN HARTFORD, (241) CONNECTICUT, DURING 1904-1905. W. R. STEINER and C. B. INGRAHAM, Jr. (of Hartford), *Amer. Journ. Med. Sc.*, March 1908.

THERE have been three distinct epidemics of cerebro-spinal meningitis in Connecticut since 1806. The first continued from 1806 to 1816, the second was in 1823, and the third in 1873.

The epidemic which furnishes the subject for this paper began in March 1904 and lasted until December 1905; 145 cases were reported, 74.48 per cent. died; 120 occurred in the first two decades of life; more are recorded from birth to five years of age than during any other period of life.

Occupation.—School children and labourers furnished most of the cases; none were reported from the well-to-do classes during this epidemic.

Etiology.—Of 55 cases lumbar punctures were performed in 51, and 43 of these showed meningococci in smears made from the centrifuged sediment; the organisms were mostly intracellular and stained by Gram's method.

Contagion.—There were but 12 possible examples of contagion during this epidemic; on the other hand, there were several families where but one case developed.

Morbid Anatomy.—Autopsies were performed upon 7 of the 37 patients that died. In the most acute case (two days) there was merely congestion of the blood vessels of the pia arachnoid. Four other cases showed, beside this congestion, a marked purulent exudate following the course of the blood vessels. In the cases of longer standing no exudate was found, but the pia was markedly thickened and injected; the exudate was more commonly found on the posterior surface of the dorsal and lumbar cord.

Onset was rapid in all but 7 of the hospital patients.

Symptoms.—Occipital headache at the onset was the most constant symptom; 27 were unconscious, 10 wildly delirious on admission; 27 showed partial stiffness of the neck, 3 marked opisthotonus; 48 out of 51 showed Kernig's sign; 8 showed absent knee-jerks, 4 exaggerated, 5 diminished, and 7 normal (others not examined). A positive Babinski reflex was never obtained; herpes was noted in about 29 per cent.; continued vomiting was present in 30 cases; 2 showed marked mental impairment.

Blood.—Examinations made in 48 patients showed an average leucocyte count of 29,555.

Urine.—Examination showed albumen, granular and hyaline casts in 28 out of the 42 examined. During convalescence the casts and albumen cleared up in those who recovered.

Types of Fever.—In 48 out of 55 the temperature charts were complete, but showed nothing typical or distinctive. They can be resolved into four groups: 1, remittent fever type (most fatal); 2, typhoid type; 3, pneumonia type; 4, ascending fever type.

Treatment.—Hot baths and packs to lessen the delirium and relieve pain, lumbar puncture to relieve pressure where extreme headache and opisthotonus existed. Antitoxin and antistreptococcus serum give little satisfaction; opium seemed to be the best sedative.

C. H. HOLMES.

EPIDEMIC CEREBRO-SPINAL MENINGITIS, etc. W. Dow,
(242) *Lancet*, March 14, 1908, p. 768.

THE clinical symptoms of the cases admitted into Belvidere Hospital, Glasgow, from May 1906 to May 1907. 183 cases, numbers of males and females practically identical. Mortality, 73 per cent. Only a few of the main points can be noted here. Onset generally sudden, headache and vomiting almost invariably, and vertigo frequently. The hæmorrhagic skin rash was seen comparatively rarely—21 per cent. of cases. When present, it has appeared most commonly on the third and fourth days, and is rapidly evanescent. Herpes was noted as often as the petechial rash.

The nervous symptoms varied greatly with the intensity and extent of the meningitis, but certain symptoms occurred in nearly all—pain, delirium, hyperæsthesia of the skin, contraction of certain muscles, exaggerated plantar reflexes, rapid emaciation and hydrocephalus in the chronic cases, convulsions at the onset in children, twitchings in adults. The eye and ear have been frequently affected. The temperature usually 102°-104°, and either rising or falling just before death; sometimes remittent. Pulse-rate varied greatly, usually much accelerated, but in a number of cases slow. Respiratory rate always accelerated at commencement, frequently out of proportion to the pulse acceleration, although no lung lesion could be detected. Various forms of nervous breathing in the acute cases. Vomiting the most prominent digestive disorder. Amount of urine much increased in the chronic cases. Arthritic pain rare, purulent arthritis only twice. A historical note shows that although sporadic cases have occurred in Scotland during the last 20 to 30 years, this is the first large epidemic.

J. H. HARVEY PIRIE.

ACUTE SYPHILITIC MENINGITIS: RECOVERY. (Meningite (243) aiguë syphilitique avec guérison.) CLAISSE et TOLTRAIN, *Bull. et Mém. de la Soc. Méd. des Hôp. de Paris*, p. 313, 1908.

A MAN, aged 34 years, of intemperate habits, who had contracted syphilis in 1898, was admitted to hospital on November 29, 1907. About six months previously he had exhibited a train of symptoms, probably due to cerebral syphilis, viz., ictus, epileptiform attacks, intense headache, and ocular palsy, associated with ideas of persecution, and attempts at homicide and suicide. These symptoms gradually passed off, and he was able to resume his work. On admission furious delirium, with terrifying visual hallucinations, were present. The next day he showed Kernig's sign, contracture of the neck muscles, ptosis, inequality of the pupils, and constipation. The history of syphilis had not yet been obtained, and the possibility of tuberculous meningitis was discussed. Lumbar puncture gave issue to a clear fluid under high pressure. Leucocytosis with predominance of lymphocytes was found. Neither the tubercle bacillus nor the treponema pallidum was present. The diagnosis of syphilis, which was supported by the discovery of cutaneous gummata, was made, biniodide injections were adopted, and 10 cc. of electrargol were injected into the spinal canal. During the next few days the meningeal symptoms were well marked, but the delirium was less violent. A second lumbar puncture was performed on December 2, and was followed by an injection of colloidal mercury into the spinal canal. The lymphocytes were now scanty, and there was a marked predominance of polynuclears. On December 4, complete brachial monoplegia, with some weakness of the lower limb on the same side, developed. In two days the paralysis diminished, but the temperature rose to 104°, and there was a return of violent delirium, visual hallucinations, and ideas of persecution. Cerebral vomiting also occurred. After another lumbar puncture, which still showed a predominance of the polynuclears, 10 cc. of colloidal mercury were injected. Improvement took place the next day. The headache diminished, and the palsy entirely disappeared. The last lumbar puncture was made on December 10th, when the cerebro-spinal fluid contained no albumen, and only a small number of cells (5-6 to the field). The lymphocytes again predominated. On December 20th the patient was discharged cured. The anatomical lesion in this case was regarded as a diffuse gummatous meningitis.

J. D. ROLLESTON.

TUBERCULOUS MENINGITIS IN INFANTS. (*Méningite tuberculeuse du nourisson.*) R. CLOT, *Thèses de Lyon*, 1906-7, No. 56.

TUBERCULOUS meningitis in infants is not rare. The diagnosis, as a rule, is very difficult, and often impossible, at the onset. Its evolution is much more rapid and its symptomatology much less complete below the age of two years than in older children. In addition to the hemiplegic and eclamptic forms described by Marfan, clinical forms exist in which the affection is manifested by vomiting only, by generalised rigidity, or by respiratory arrhythmia and somnolence. Often there is an unaccountable progressive emaciation. The temperature may not be raised until the terminal convulsions. Lumbar puncture is not always of help in diagnosis. The difficulty will often be solved by the effects of treatment. The digestive disturbances of tuberculous meningitis are absolutely proof against all treatment, whereas gastro-intestinal troubles which simulate tuberculous meningitis may yield rapidly to appropriate measures. Syphilitic meningitis runs a much longer course than tuberculous meningitis, and the history and other evidences of syphilis will here be a guide. Acute meningitis due to other causes is very rare in infants.

The thesis concludes with the record of twelve personal cases in infants aged from four to twenty-four months.

J. D. ROLLESTON.

DISSOCIATION OF THE COLOUR-SENSE THROUGH FOCAL (245) BRAIN DISEASE. (*Abspaltung des Farbensinnes durch Herderkrankung des Gehirns.*) M. LEWANDOWSKY (of Berlin), *Berl. klin. Wochenschr.*, No. 45, 1907.

THE disorders of the colour-sense which occur in brain disease are—true colour-blindness (total or partial, limited to the absence of red-green), amnesic colour-blindness (Wilbrand), i.e. a sensory aphasia limited to names of colours. The author had under observation a man of fifty, who suddenly developed a typical sensory aphasia (Wernicke); motor symptoms were absent, but after the disappearance of the sensory aphasia there remained a sub-cortical alexia. There was obviously a focus in the anterior region of the left occipital lobe, probably in the neighbourhood of the angular gyrus. The patient had right-sided hemianopia. The colour-sense was impaired; the patient could neither name the colours in Holmgren's wool test, nor could he pick out colours named. He could not name the colour of familiar objects, although no speech disorder was to blame for this. He failed with black

and white as well as with the colours of the spectrum; he distinguished dark and light. There was neither congenital nor acquired colour-blindness; examination of the colour-sense by the Helmholtz apparatus disclosed no defect; his memory for colours was intact. The author assumes that in the brain of the patient the colour-sense was dissociated from the perceptions and concepts of forms and objects, that it was isolated and could not be associated with the sense of light and of form. The colour-centre on the left was destroyed, the right preserved; the association of the colour-sense not only of the left retinal fields, but of the unimpaired right fields with the other elements of optic perception, took place in the brain of the patient only with the assistance of the colour-centre of the left hemisphere, and after its destruction the association disappeared. The fact that the colour-sense was unimpaired was explained by the intactness of the right-sided colour-centre. The author doubts whether in all men the association of colours with the other optic perceptions takes place in the left hemisphere. K. STEINDORFF (C.g.B.).

RESEARCHES ON TUBEROSE SCLEROSIS. (*Recherches sur la*
(246) *Sclérose Tubéreuse.*) CH. DE MONTET, *L'Encéphale*, Feb. 1908.

THE author gives a detailed description of the histological features found in the brain of a male epileptic idiot, aged 12, who died, as is usually the case in tuberosc sclerosis, from pulmonary tuberculosis. In one kidney there was a small round growth, which proved to be a hypernephron. He regards the giant cells so characteristic of this affection as aberrant nerve cells, which have no connection with the neuroglial fibres. His conclusion, agreeing with Geitlin's, is that the condition is essentially due to an alteration of the neuroblasts, which, according as the interaction of other special factors (degrees of alteration, etc.) varies, results in a diffuse histioatypism or true teratoid tumours. The relative frequency of tumours in other organs, such as rhabdomyomes and hypernephrons, is in favour of this theory. The affection commences probably relatively late in the foetus, as is indicated by the generally good configuration of the convolutions. Concerning its etiology all he has to say is that syphilis may be almost certainly excluded.

He gives a list of some twenty papers by continental writers on the subject, but makes no mention of A. W. Campbell's valuable article in *Brain*, 1905.

Campbell describes, probably for the first time, peculiar gland-like structures in the cortex, and, according to him, the disease is the outcome of some evolutionary aberration or disturbance, arising

late in foetal life, affecting the endothelium of blood vessels or lymphatics, and resulting in structural hyperplasia and heterotopism.
JOHN TURNER.

MULTIPLE SYPHILITIC LESIONS. TABES, GENERAL PARALYSIS, AND AORTIC REGURGITATION. (Lésions syphilitiques multiples. Tabes, paralysie générale et insuffisance aortique.) DEBOVE, *Gaz. des Hôp.*, févr. 20, 1908, p. 243.

THIS is the report of a clinical lecture specially destined to call attention to the dangers that may at any time occur to an individual who has once had syphilis. Debove points out that even the most thorough and prolonged treatment may be followed by an outbreak of tertiary syphilis or parasyphilis, and asserts that the spirochæte probably survives in the body throughout the individual's life. He then demonstrated a typical case showing the above-mentioned three complications. The patient, a man of forty, had had syphilis at twenty, and had no after manifestations of it until these occurred. He refers to the prophylactic action of mercury administered some hours, and atoxyl some days, after exposure to infection.
ERNEST JONES.

THE SIDE AFFECTED BY HYSTERICAL HEMIPLEGIA. (Le côté de l'hémiplégie hystérique.) ERNEST JONES, *Rev. Neurol.*, mars 15, 1908, No. 5.

THE universally accepted opinion that hysterical hemiplegia affects the left side three times as often as the right is based entirely on Briquet's cases, examined before 1859, and has never been investigated since. The author has collected the cases published since 1880, full references to which are given, since which date the diagnosis of hysterical hemiplegia has been more secure. The results are given in the following table:—

	Number.	Percentage on right.	Percentage on left.
Briquet, 1859	60	23·3	76·6
Cases since 1880	277	54·2	45·8

The author concludes that there is no evidence that hysterical hemiplegia affects one side more than the other, and that the question of the side of a hemiplegia is of no value in relation to the diagnosis of its origin.
AUTHOR'S ABSTRACT.

THE PRECISE DIAGNOSTIC VALUE OF ALLOCHIRIA. ERNEST (249) JONES, *Brain*, 1907, Vol. xxx., pp. 490-533

THE author first reviewed 76 cases published under the name of allochiria, 12 being the largest number previously collected, and considers that only 29 of these are true cases of this condition. In 26 of the 29 there was no reason to suppose that any affection other than hysteria was present, and in the remaining three hysteria was present in addition to an organic affection. On the basis of personal observations, to be detailed later, he describes the various manifestations of the condition and discusses the previous hypothesis concerning it. The article is summarised in the following conclusions:—

1. Under the name of allochiria two fundamentally different conditions have hitherto been confused. A patient's mistake in determining the side of a stimulus may be—(i) part of a general defect in localisation—alloæsthesia; or (ii) a specific defect independent of any error in localisation—dyschiria. The name allochiria has further been incorrectly applied, as in the terms electromotor and reflex allochiria, to symptoms which are in no way related to either of these conditions.

2. Dyschiria may be defined as a state in which there is constantly either ignorance or error in the patient's mind as to the side of given stimuli, quite independent of any defect in sensorial acuity or in the power of localisation. This corresponds closely with the definition of allochiria given by Obersteiner, though he did not distinguish the condition from alloæsthesia.

3. There are three stages of dyschiria: achiria, in which the patient has no knowledge as to the side of the stimulus; allochiria, in which he refers it to the corresponding point on the opposite side; and synchiria, in which he refers it to both sides. There are three sub-varieties of the latter.

4. All writers subsequent to Obersteiner have abstracted one feature from his definition, namely, the reference of the stimulus to the opposite side, and have used it to define allochiria. It is suggested that the term allochiria be always used in this its current sense, with, however, the important proviso insisted on by Obersteiner, that the symptom is independent of any defect in sensorial acuity or in the power of localisation. The significance of this proviso has been entirely overlooked hitherto, and even Obersteiner did not recognise that a direct corollary of it is the separation of the alloæsthetic from the allochiric error. There are seven precise clinical features that enable a differential diagnosis between alloæsthesia and allochiria to be made with certainty.

5. Alloæsthesia is adequately explained by the Head-Spearman hypothesis that it is due to a defect in afferent excitations, particu-

larly those of the "articular" type. It occurs in both organic and functional disease, perhaps most often in tabes.

6. Dyschiric manifestations may be general in distribution or may relate only to certain segments of the body. There are characteristic introspective, motor, and sensory manifestations of each member of the group. The last-mentioned may occur in connection with all varieties of stimuli or with only some. The motor allochiria has been unnecessarily termed "allokinesia." Sensation resulting from stimulation of a dyschiric part has six peculiar attributes, here grouped under the designation "phriktopathic."

7. There have been three explanations of allochiria hitherto offered. The Fischer-Hammond hypothesis is throughout contradicted by the facts and should be entirely discarded. The Head-Spearman hypothesis refers to alloesthesia only, and has no relation to allochiria. The Head-Tanet hypothesis is not borne out by the observations on which this paper is based, which seem to demonstrate that allochiria is independent of any defect in sensorial acuity.

8. Dyschiria is due to psychical disaggregation, and is distinctive of the form of disaggregation characteristic of hysteria. It is primarily an affection of the feeling of "sidedness" (the chirognostic sense).

9. Of the three stages of dyschiria, achiria represents the most severe grade of disaggregation and synchiria the least. These two are essentially transitional forms. Allochiria, on the other hand, is a stable condition which may be present for years.

10. A number of fallacies in diagnosis are here indicated, and especial attention drawn to the close resemblance between unilateral achiria and hemiplegia, particularly hysterical hemiplegia.

AUTHOR'S ABSTRACT.

A FATAL CASE OF PONTILE HEMORRHAGE, WITH AUTOPSY.

(250) T. DILLER (of Pittsburg), *Amer. Journ. Med. Sc.*, March 1908.

REPORTED cases of pontile hemorrhage are comparatively rare; about sixty-seven cases had been reported up to 1890. Since that time perhaps the most valuable contributions have been made by Dana, Bode, Larcher, Glasser, and Deshusses.

Among the most important symptoms of the disease are vomiting, irregular respirations, polyuria, single or double hemiplegia, paralysis of the lower face and of the tongue and larynx (Guillain), elevation of temperature (Erb), prodromal headaches, malaise, sudden development of profound coma, and death in from six to twenty-four hours (Dana).

Diller's case was that of a woman of sixty-three years, who had been in the City Home continuously for thirty-four years. She

developed a sudden attack of purging and vomiting, respirations hurried and of a stertorous character, temperature 99°; later double hemiplegia and myosis occurred, and death took place in thirty hours.

Autopsy showed basal vessels in a state of advanced degeneration, a firmly coagulated hemorrhage extended over the pons and slightly over the medulla, the exits of all the cranial nerves were involved in the clot; the hemorrhage was thought to have come from the basilar artery.

The diagnosis in this case was ventured, on the basis of double hemiplegia, myosis, and the suddenness of onset with vomiting.

C. H. HOLMES.

**DOUBLE PERSONALITY FOLLOWING HÆMORRHAGE. (Dé-
(251) doublement de la personnalité à la suite d'hémorrhagie.)**

PAILHAS, *L'Encéph.*, Fév. 1908, p. 139.

IN a few lines are mentioned two cases in which a severe hæmorrhage—one gastric, the other intestinal—was followed by the hallucination of consisting of two bodies. For some inexplicable reason the author calls these cases of double personality, and adds some hypothetical conjectures about excitation of the cœnæsthetic centres. He adds that his two cases seem to show that this cœnæsthetic projection occurs more often on the right side!

ERNEST JONES.

MENTAL DISTURBANCES IN VASOMOTOR NEUROSES.

(252) (*Psychische Störungen bei der vasomotorischen Neurose.*)

ROSENFELD, *Centralbl. f. Nervenheilk. u. Psychiatr.*, Bd. xxix., S. 137-159.

AFTER some general remarks on the principles of classification of the psychoses, Rosenfeld continues the discussion begun in two former contributions as to the possibility of defining a group of mental affections accompanied by vaso-motor symptoms. He adds eight to his five previously described cases. The neurological symptoms were acrocyanosis, colour changes, dermographia, disagreeable cardiac sensations, bradycardia, giddiness, especially on standing, and marked sweating, there being no evidence of organic disease of the nervous system. The mental symptoms were exaggerated anxiety for relatives, feeling of inadequacy, self-reproach, phobias, emotivity, with absence of psychomotor inhibition and retained insight into the condition present. The differential diagnosis is difficult, especially from hysteria. Relapses are frequent, but the ultimate outlook is good. The author unconvincingly seeks to

establish here a separate clinical entity, mainly on the grounds of the typical course of the affection and the difficulty of otherwise grouping it.
ERNEST JONES.

HYSTERIA IN CHILDREN. G. E. PRICE, *Arch. of Pediatrics*, Feb. (253) 1908, p. 95.

A BRIEF report of four cases. One, a girl of 10, with hysterical tremor in the arm; tremor appeared in a leg as the result of suggestion. Definite hysterical stigmata. Case ii., a boy of 13, with spasmodic attacks of pain in the hip-joint ever since a fall eleven months before. No organic lesion. Areas of hypæsthesia and hypalgesia distributed irregularly over the limbs. Case iii., a girl of 13, was of the psychic type. Very emotional, but no motor or subjective sensory symptoms. Bad neuropathic family history. Case iv., a girl of 15, was the subject of attacks of hysterical dyspnoea.
J. H. HARVEY PIRIE.

CONTRIBUTION TO THE CYTOLOGICAL STUDY OF THE
(254) **CEREBRO-SPINAL FLUID.** (Beiträge zum Zellstudium der Cerebrospinalflüssigkeit.) M. PAPPENHEIM, *Zeitschr. f. Heilk.*, H. 10, 1907.

THE author has made the interesting discovery that every cerebro-spinal fluid exercises a deleterious influence on white blood corpuscles; this quality is especially well marked in the fluid of general paralytics, and heating the fluid to 56° destroys this action. It is, therefore, advisable to add formalin to the cerebro-spinal fluid soon after its withdrawal. Many peculiar elements described in the cerebro-spinal fluid are merely degenerated leucocytes, which have been acted on by the leucotoxin. The author has especially studied the elements described by Sabrazès and Muratet, under the name of "hæmatomacrophage" elements. Their presence may be due to cerebral hæmorrhage dating back about a week; yellow colouration points to an older hæmorrhage, perhaps due to a previous puncture. The author shows that the "hæmatomacrophage" elements may have a similar cause, for in one of his cases he found these cells in the dura mater at the site of a previous puncture.
H. HIRSCHFELD (C.G.B.).

LYMPHOCYTOSIS OF THE CEREBRO-SPINAL FLUID IN LUES
(255) **HEREDITARIA TARDA.** (Lymphocytose des Liquor cerebrospinalis bei Lues hereditaria tarda.) W. KRETSCHMER (of Wiesbaden), *Deutsche med. Wochenschr.*, No. 46, 1907.

IN the cerebro-spinal fluid of infants and young children with hereditary syphilis Tobler has demonstrated a lymphocytosis.

The author has worked on the same lines at late hereditary syphilis, but has comparatively little material. In two cases he found a marked lymphocytosis; further investigation along these lines is required.

H. HIRSCHFELD (C.g.B.).

**THE UNSUCCESSFUL SEARCH FOR CHOLIN IN THE CEREBRO-
(256) SPINAL FLUID.** (Ueber den angeblichen Befund von Cholin
in der Lumbalfüssigkeit.) M. KAUFFMANN, *Neur. Centralbl.*,
16th March 1908.

KAUFFMANN, of the clinic for nervous diseases at Halle, has investigated the cerebro-spinal fluid obtained by lumbar puncture with special reference to the existence in it of cholin, as was affirmed by Moll and Halliburton in 1899. He has failed to find it in any appreciable quantity, or even with certainty at all, though a litre of fluid was obtained for examination. There is a base present, but its nature is still a matter of doubt. What alone appears certain is that it is not cholin. He also failed to find pyrocatechin, which Halliburton states to be present. The cases examined were general paralytics and epileptics, in whom there is thought to be an unusually rapid breaking down of nervous material. His conclusion is that if cholin is present in the cerebro-spinal fluid of the insane, it is so in such small quantity that it cannot be chemically identified. It has therefore no pathological significance.

JAS. MIDDLEMASS.

SERUM DIAGNOSIS OF SYPHILIS. (Serodiagnostik der Syphilis.)
(257) F. PLAUT (of Munich), *Zentralbl. f. Nerven- u. Psych.*, Ht. 8,
1908.

IN the hæmolytic experiment with the original technique of Wassermann (who employed watery extract of syphilitic organs as antigen), the positive reaction of the blood serum in undoubted cases of syphilis has been present in 80-90 per cent. The reaction is specific, and is never present in a non-syphilitic individual; it enables us to diagnose the constitutional disorder, but not the organ affected. Examination of the cerebro-spinal fluid by this method sometimes enables us to make a diagnosis of the organ affected.

In twenty-five cases of syphilis, without involvement of the central nervous system, the author never found an anti-body in the cerebro-spinal fluid, while the serum, as a rule, gave a positive reaction. Even in actual syphilis of the central nervous system the cerebro-spinal fluid is very frequently free from syphilitic anti-bodies. On the other hand, in all but one of ninety-five cases of general paralysis examined by the author, the cerebro-

spinal fluid gave a positive result; the serum, in every case of general paralysis examined, gave a positive reaction. In cases of cerebral syphilis the serum was usually positive, the cerebro-spinal fluid usually negative; in tabes the cerebro-spinal fluid gave a positive reaction in 70-80 per cent. of the cases. The author emphasizes the value of the method as an aid in separating from other groups those cases of congenital or early acquired mental enfeeblement which arise on a syphilitic basis; it may also help in the differentiation of arterio-sclerosis on a syphilitic basis from that with different etiology.

As to the nature of the antigen, views have considerably changed. Wassermann and his collaborators observed the specific interference with hæmolysis only with extract of a syphilitic organ; Marie and Levaditi observed the same phenomena sometimes with extract of normal liver in concentrated solution, and the author confirms their results. It was then found that the antigen principle could be extracted with alcohol, and that here, too, certain normal organs would serve the purpose. The antigen, therefore, seemed to belong, not to the albumen group, but to the lipid substances; Porges accordingly substituted lecithin for organic extract in the hæmolytic experiment, and in some cases obtained positive results.

Various attempts have been made to arrive at a simpler technique of serum diagnosis by means of precipitation. For this purpose Porges employed equal quantities (.2 c.cm.) of a .2 per cent. suspension of lecithin in .5 per cent. carbolic normal saline solution and of syphilitic sera. The results were positive, but by no means absolutely specific.

The author sums up the various methods and their various advantages.

A. Method of Binding of Complement.

1. The original method of Wassermann, Neisser, and Bruck, of binding complement by means of a watery extract of a syphilitic organ is even to-day the most reliable.

2. One only rarely finds watery extracts of non-syphilitic organs which have a specific action in hindering hæmolysis, and these extracts are of less value than the afore-mentioned.

3. The alcoholic extracts of syphilitic and normal organs give a reaction which is specific for syphilis, but not with the same regularity or intensity as the first-mentioned.

4. Lecithin cannot be recommended as a substitute for extract of a syphilitic organ in the hæmolytic experiment.

B. Precipitation Method.

1. Fornet's method is of no use.

2. The phenomenon observed by Michaelis, that precipitation

occurred on mixing watery and syphilitic extracts with sera, is of little practical use, as it is of rare occurrence.

3. The method of Porges-Meier, who employed lecithin, is not sufficiently specific to be of much practical use.

4. The phenomenon observed by Klausner (precipitation of globulin on mixing syphilitic sera with distilled water) is not sufficiently specific for syphilis. C. MACFIE CAMPBELL

CONGENITAL SYPHILIS. A. E. WYNNE, *Dub. Journ. of Med. Sci.*, (258) March 1908, p. 191.

NOTES of a case, probably of diffuse syphilitic sclerosis, with dementia, due to congenital disease, in a lad of 17.

J. H. HARVEY PIRIE.

ACUTE RETROBULBAR NEURITIS AND MULTIPLE SCLEROSIS.
(259) (*Neuritis retrobulbaris acuta und multiple Sklerose.*) B. FLEISCHER, *Klin. Monatsbl. f. Augenheilk.*, Feb. 1908.

THE significance of retrobulbar neuritis in the diagnosis of multiple sclerosis has already been investigated by Uhthoff and others. The real ætiology of this "idiopathic" neuritis has always been obscure, and it was a desire to determine whether disseminated sclerosis had any causal relationship with it that led the author to investigate the after-history of a number of cases of acute retrobulbar neuritis, as well as certain cases of acute optic neuritis (papillitis) whose clinical course agreed with that of the retrobulbar variety.

Patients who had been previously treated in the Tübingen clinic for these conditions were written to, and such of them as were suffering from any disturbances of their general condition were asked to attend at the clinic; a thorough examination of the nervous system was then made in every case.

Thirty cases of retrobulbar neuritis were examined, only those who had shown the condition in its typical acute "idiopathic" form being taken into account; cases which depended on direct extension of inflammation from the orbit, nasal cavities, or neighbouring parts were also excluded. Twelve cases of marked papillitis were also examined. The individual histories are all given, in an abbreviated form. Most of the patients were under thirty-one years of age.

The result of the investigations was to show that by far the larger number of these cases did, in point of fact, suffer from multiple sclerosis.

For a diagnosis of this latter condition we do not nowadays

need to wait for a full development of Charcot's classical picture (intention-tremors, nystagmus, and scanning speech); certain less pronounced symptoms, as laid down by the researches of Oppenheim and Uhthoff, enable us to establish a no less reliable diagnosis at a much earlier period; specially characteristic are the occurrence of very transient palsies, and of a peculiar type of paræsthesia.

It is specially to be noted that a long time may elapse between the appearance of the retrobulbar neuritis and that of the other symptoms of multiple sclerosis, hence the great importance of the former condition as an early symptom in the diagnosis.

As regards the twelve cases examined, in which well-marked optic neuritis existed, here, too, it was demonstrated that almost half the number were suffering from multiple sclerosis.

The fact was thus established that acute retrobulbar neuritis represents a (mostly) initial symptom of multiple sclerosis. Of the cases of more marked involvement of the papilla (obvious papillitis), half became the subjects of multiple sclerosis; it is possible that the percentage of this latter group may be even larger.

This confirms the observations of the neurologists that visual disturbances of the nature of acute retrobulbar neuritis are not uncommon in the anamnesis of disseminated sclerosis cases; it also confirms Peter's view as to the ætiological importance of multiple sclerosis in cases of retrobulbar neuritis, and amplifies the experiences of Marcus Gunn, who, in 1905, found that of 223 cases of acute retro-ocular neuritis in which the optic nerve was primarily affected, at least 51 were demonstrably caused by multiple sclerosis (*Ophth. Review*, vol. xxiv., p. 285).

Thus in cases of acute retrobulbar neuritis in young adults, where no obvious cause, such as inflammatory extension, intoxications, or the like, can be demonstrated, one may with considerable probability of correctness make a diagnosis of incipient multiple sclerosis.

ARTHUR J. BROCK.

THE PHYSIOLOGY AND PATHOLOGY OF THE PUPILLARY

(260) **MOVEMENTS.** (*Die Physiologie und Pathologie der Pupillenbewegungen.*) O. BUMKE (of Freiburg), *Med. Klin.*, 1907. No. 41.

THE width and movements of the pupils do not depend on elastic phenomena nor on variations in blood-pressure, but on the tonicity of the antagonistic muscles of the iris; these are the sphincter and the dilator of the pupil, the former supplied by the third nerve, the latter as well as the muscle of Müller (symptom-complex of

Horner) being supplied by the sympathetic. Cocaine stimulates the dilator, fails to act therefore in sympathetic paresis; eserine stimulates the sphincter, atropine and its derivatives paralyse the sphincter. Convergence, accommodation, and narrowing of the pupil are associated movements which depend on a central impulse. They are of less diagnostic importance than the light reflex, the bilateral occurrence of which on illumination of one eye (consensual reaction) depends on the decussation of the optic nerves in the chiasma, and the connection of each sphincter nucleus with the retina of its own eye.

The disorders of innervation of the pupils include amaurotic rigidity, absolute rigidity, paralysis of the sphincter, which occurs as one element of a total or partial paralysis of the third. The more or less complete paresis of the third nerve has several causes, and is a nuclear affection, just as paresis of the internal muscles of the eye (internal ophthalmoplegia); uncomplicated absolute immobility is almost always due to syphilis. True reflex immobility (Argyll Robertson's symptom), in which the sensory centripetal and the motor centrifugal branch of the reflex arc are intact, but the light reflex eliminated, while the reaction on convergence is preserved, represents a central disorder which is situated between that part of the oculomotor nucleus presiding over the sphincter iridis, and the termination of the optic nerve in the external geniculate body. It is rare, because it depends on the destruction of a few isolated fibres, and is symptomatic of metasymphilitic tabes or general paralysis; in extremely rare cases it occurs as an isolated nervous sequel of syphilis.

It is necessary to guard against confusing this symptom with paresis of the sphincter. In 20 per cent. of cases of tabes or general paralysis there are no disorders of pupillary innervation present.

The sign of Argyll Robertson almost always points to involvement of the posterior columns, and therefore to be specifically tabetic; these changes, as well as rigidity of the pupil, are almost always present in the terminal stage of general paralysis. No conclusions as to the pathological anatomy of reflex immobility of the pupil can be drawn from these observations (degeneration of certain fibres in the cervical cord, reflex centres in the medulla). It has not yet been possible to demonstrate any characteristic changes in the region between the geniculate body and the sphincter nucleus.

K. STEINDORFF (C.g.B.).

ON THE DIAGNOSTIC APPLICATION OF ESERIN IN PUPIL-
(261) LARY DISORDERS. (Zur diagnostischen Verwertung des
 Eserins bei Pupillenstörungen.) F. F. KRUSIUS (of Marburg),
Zeitschr. f. Augenh., Bd. xviii., H. 5, 1907.

THE author gives in tabular form the results of his examination of cases of paresis or paralysis of the sphincter pupillæ, and concludes that eserine does not help us in determining whether in absolute immobility the central or peripheral neurone is affected—that is, whether the lesion is anterior or posterior to the ciliary ganglion.
 K. STEINDORFF (C.G.B.).

ON A PECULIAR PUPILLARY PHENOMENON; BEING AT THE
(262) SAME TIME A CONTRIBUTION TO THE QUESTION OF
HYSTERICAL IMMOBILITY OF THE PUPIL. (Ueber ein
 eigenartiges Pupillenphänomen; zugleich ein Beitrag zur
 Frage der hysterischen Pupillenstarre.) REDLICH, *Deutsche*
med. Wchnschr., February, p. 313.

THE basis of the author's observations was the case of a female epileptic, 33 years of age, who was also the subject of hysterical attacks; these took place characteristically during the doctor's visit, and occurred as long as notice was taken of them. The patient twisted herself now to one side, now to the other, threw her head back, cried out loudly and continuously, and threw her hands about. The attacks were short, but followed each other quickly. Consciousness was not lost; patient responded to simple requests, as, *e.g.*, to show her tongue, etc. During these attacks the pupils were circular, widely dilated, and did not usually react to light (a small pocket electric lamp was used). This stage of dilated and irresponsive pupil was often preceded by one in which the pupils showed a more than medium dilatation, were obliquely oval and irregular in shape, and contracted imperfectly to light. Between the individual attacks, and apart from them, the pupils were of medium width, and reacted promptly to light-stimuli as well as to convergence and accommodation. The fundus was normal; vision good.

Careful observation proved that an exactly similar condition of the pupil (*viz.*, dilatation and immobility) could be artificially produced by causing the patient to go through, of her own will, movements similar to the involuntary ones of the fit—*i.e.* by making her cry out, throw her muscles into strong contraction, etc. Here, too, the condition was often preceded by a stage in which the pupil became oval and showed an imperfect reaction to light. It was necessary that the muscular effort should be strong,

and, above all, persistent; the least slackening in effort was followed by some contraction of the pupil and by its beginning to react to light. The author was inclined to think that this phenomenon occurred most readily with certain special movements—*e.g.* when the patient pressed an assistant's hands crosswise with all her force, her head being bent back and her cervical muscles powerfully contracted; at the same time the breath was held and the face became congested.

That strong muscular contractions normally produce dilatation of the pupil is a fact of common observation, but the dilatation is not usually great, and the pupillary light-reaction is little, if at all, altered.

The reaction in the present case was thus simply an extreme exaggeration of the normal.

Out of various possible explanations of this phenomenon which suggest themselves, the author prefers the view that it depends on strong muscular contractions, especially on direct irritation of the cervical sympathetic by the muscles of the neck.

Irritation of the sympathetic (*i.e.* spasm of the dilator pupillæ) is not in itself sufficient to produce maximal dilatation and absolute failure of response by the pupil to light-stimuli; paralysis, or inhibition of the sphincter, is required besides. In the case under consideration, however, and in other similar cases, a slight reaction was retained, *viz.*, that to convergence; thus the majority can be explained by a mere spasm of the dilator.

This view may furnish us with an explanation of the abnormal width of pupil so common in hysteria and epilepsy; it will not, however, explain all hysterical disturbances of the pupil, and only holds good of the convulsive stage of the attack.

Moreover, quite apart from the attacks themselves, we may get various disturbances of the pupil in epilepsy, such as total immobility, or contraction and immobility combined; the latter is a case of miosis and immobility depending on spasm of convergence. These various pupillary disturbances can be explained as exaggerations of normal reactions, dependent on some neurosis (perhaps on abnormally increased irritability); *e.g.*, the contraction of the pupil which always accompanies convergence may become exaggerated into miosis and failure of response to light-stimuli.

ARTHUR J. BROCK.

THE EYE-MOVEMENTS IN CEREBELLAR IRRITATION. (Über (263) die Augenbewegungen bei Kleinhirnreizung.) A. LOURIE, *Neurol. Centralbl.*, Feb. 1, 1908, p. 102.

THE author maintains that he has demonstrated that the cerebellum has no influence upon the movements of the eye-balls.

He concludes from his experiments that the surface of the cerebellum does not contain centres for the movements of the eyes and facial muscles, but he does not appear to have made observations upon the effects of irritating the intrinsic nuclei of the cerebellum, so that he is hardly justified in saying that there are no special centres in the region of the cerebellum for these movements.
ALEXANDER BRUCE.

THE ETIOLOGY OF ABDUCENS PARALYSIS, ESPECIALLY OF (264) ISOLATED PARALYSIS. (Zur Aetiologie der Abducenslähmung, besonders der isolierten Lähmung.) KÖLLNER, D. *med. Wchnschr.*, Jan. 16 u. 23, 1908.

THE writer refers to the relatively great frequency of paralysis of the sixth nerve. Among the etiological factors he includes alcoholism, arterio-sclerosis, lead paralysis, intra-spinal injection of stovaine, etc., for anæsthesia, malaria, influenza, erysipelas, albuminuria, diabetes, various diseases of the brain and spinal cord, etc.
ALEXANDER BRUCE.

OCULO-MOTOR PARALYSIS WITHOUT INVOLVEMENT OF (265) THE INTERNAL MUSCLES IN PERIPHERAL LESIONS. (Oculomotoriuslähmung ohne Beteiligung der Binnemuskeln bei peripheren Läsionen.) E. FUCHS, *Arb. a. d. Neurol. Instit. a. d. Wien. Univ.*, Bd. xv., T. 1, 1907, p. 1.

It was formerly thought that in a purely external ophthalmoplegia the origin could not be in the peripheral nerves, but must be in the oculo-motor nuclei or in the intra-cerebral root region. But the number of cases is increasing in which there has been a peripheral lesion with a purely external ophthalmoplegia, either unilateral or more rarely bilateral, or where the oculo-motor nerve has been alone injured, the interior muscles of the eye having escaped the paralysis. The author gives references to eighteen such cases, in six of which there had been a sectio, showing as lesions compression atrophy, primary atrophy of the nerve-stem, and inflammation, and he describes five clinical observations of his own.

From study of all these cases he concludes that a purely external ophthalmoplegia depends upon no special kind of anatomical lesion, but may be observed in trauma, inflammation, simple atrophy and atrophy from compression. He thinks that the escape of the internal muscles is not so much due to their central position as to their slighter degree of vulnerability. In support of this view he refers to the facts that in neuritic optic

atrophy, while the sensibility to light is lost, yet the light-reaction of the pupils is retained owing to greater resistive power of the fibres concerned, and that in compression of the intra-cranial or orbital part of the optic nerve a central scotoma due to a lesion of the papillo-macular bundle of the optic nerve may be the first symptom, probably on account of its special vulnerability.

ALEXANDER BRUCE.

CONJUGATE DEVIATION OF THE EYES AND HEAD. (*Dévi-
ation* (266) *conjuguée des yeux et de la tête.*) A. DEBRAY, *Journ. de
Neurol.*, oct. 5 et 20, 1907.

Two main varieties of conjugate deviation of eyes and head are described:—(1) That in which the head deviates in the same direction as the eyes, and (2) that in which the eyes and head are turned laterally in opposite directions. The author discusses the parts played by the various afferent and efferent tracts in the pons, cerebellum, superior peduncles, central nuclei and internal capsule, and cerebral convolutions. He goes into the details of the various theories with regard to the mechanism of the movements, but does not materially add to our knowledge of the subject.

ALEXANDER BRUCE.

ON THE INFLUENCE OF ROTATORY MOVEMENTS ON NYSTAG-
(267) **MUS.** (*Ueber den Einfluss von Drehbewegungen um die
vertikale Körperachse auf dem Nystagmus.*) CASSIRER and
LOESER, *Neurologisches Centralbl.*, 1908, p. 252.

THE observations here recorded were undertaken with the object of testing the functional activity of the vestibular nerve. In many respects they are similar to the well-known observations of Bárány and of Wanner.

The chief point emphasised in this paper is the fact that rotation of the body around its own long axis, by seating the patient on a turn-table, produces well-marked effects on any pre-existing nystagmus. The patient is first carefully observed to notice whether ocular movements in any direction, the head remaining fixed, produce nystagmus. The patient is then rotated six to ten times on a revolving stool, and immediately afterwards the nystagmus is examined afresh. The writers distinguish between undulating nystagmus, in which both phases of the nystagmus are equal, both in range and in speed, and rhythmic nystagmus, in which both movements are of equal range, but the one is slow and the other fast.

The conclusions to which they come are as follows:—Horizontal

nystagmus on lateral movement of the eyes can be made to disappear on looking to one side, after rotation around the long axis of the body. After such a rotation, nystagmus, on looking towards the direction of rotation, disappears, whilst on looking in the reverse direction, the nystagmus is intensified. Even in a normal individual, after such rotation, nystagmus appears on looking in the opposite direction from that of the previous rotation. The nystagmus is attributed by them to labyrinthine stimulation, from movement in the endolymph.

PURVES STEWART.

THE SPINAL CORD IN CASES OF ABSENCE OF THE PUPIL-

(268) **LARY LIGHT REFLEX.** (*Das Verhalten des Rückenmarkes bei reflektorischer Pupillenstarre.*) H. WUNDERLICH, Inaug.-Diss., Würzburg, 1907, pp. 28.

ON the basis of the examination of the cord in fifteen general paralytics the author concludes: "There appears to be a constant connection between degeneration of the posterior columns and reflex immobility of the pupil." The posterior columns of the upper part of the cervical cord (especially one definite region—namely, the ventral part of Bechterew's intermediate zone) were only degenerated in cases where clinically there had been long-standing loss of the light reflex; they were always intact in cases where the pupils were normal or showed only slightly impaired reaction.

F. LOEB (C.G.B.).

TOXIC AMBLYOPIA. (*Amblyopie toxique par l'alcool et le tabac.*)

(269) POULARD, *Prog. Méd.*, March 7, 1908.

THIS condition, so frequent in drinkers and smokers, is easily recognised, and the unjustifiable carelessness with which so many medical men regard its existence cannot be set down to any difficulty in diagnosis.

The normal retina is divided, both from an anatomical and a physiological point of view, into two parts—(a) the small, highly-sensitive "central retina" (macula); (b) the much larger and much less sensitive peripheral portion.

The one constant and unfailing symptom of toxic amblyopia (alcoholic or tabetic) is the *loss of central vision, with retention of peripheral vision*; from this alone a sure diagnosis can be made, and it is unnecessary to investigate other less important symptoms, of which many exist.

This change in central vision is shown by an inability to see the fixation object clearly, and, at the same time, to distinguish colours, especially green and red. The eye cannot see the colour

of the fixation object, but can do so as soon as that object passes into the periphery of the field of vision. There may be a central scotoma for green, red, etc., or for all colours, or the scotoma may be *absolute*, when there is blindness even for white light.

To examine the central vision of a patient, make him close one eye and hold up before him, at a distance of about 50 cm., a large blackened surface of cardboard or metal, in the centre of which there is a small square or round aperture; let him look through this with his open eye, and name the colours of a series of papers or other objects which are passed behind the orifice. We begin with green, as this is the first colour to disappear; the existence of a central scotoma for green is pathognomonic of alcoholic amblyopia; moreover, this type is characterised by being *bilateral*. To absolutely confirm this diagnosis, make the patient look straight at your finger, and, while he does so, move the blackened surface a little outside the fixation point; he will at once be able to distinguish the coloured or white paper through the hole, though he could not do so when looking straight at it.

Toxic amblyopia proves specially trying for those who read much, for watchmakers, etc., but in the case of outdoor workers, coachmen and the like, it gives less trouble, as in these cases the integrity of peripheral vision is of primary importance.

Differential diagnosis between tobacco and alcoholic amblyopia is based entirely on the anamnesis, but the two factors are often combined.

The condition is practically always slow and progressive, and after the poison has been removed, retrogression is on the same lines. A complete cure can only be hoped for when the amblyopia is not far advanced, and, in any case, the affection is slow to yield to treatment, which consists, essentially, in removal of the cause.

ARTHUR J. BROCK.

GALVANIC REACTIONS OF THE AUDITORY NERVE. (Zur (270) Klinik der galvanischen Akustikusreaktion.) ALICE MACKENZIE, *Wien. klin. Wochenschr.*, Nr. 11, 1908.

BRENNER states that the normal reaction of the auditory nerve is as follows:—With the kathode in the meatus closure of the current produces a marked sensation of sound, which remains during the passage of the current and ceases with the opening: if the anode be in the meatus the result is negative, except that a slight noise is heard at opening. Other observers have not confirmed these results. Pollack and Gärtner only occasionally obtained a reaction with currents of six milliamperes in normal cases, while patients suffering from ear disease reacted to ONE or TWO milliamperes. In the present investigation one electrode was applied over the tragus

and the other was held in the hand. The patients were both suffering from extreme degrees of deafness and tinnitus: the strength of the galvanic current was increased until the patient noticed a distinct augmentation of his subjective noises. The amount of current required was noted, then reduced to nothing, and the poles reversed. Four milliamperes was found to be the best strength of current for testing the reactions of the nerve at closure and opening. The first case, aged 22, had been deaf for twelve years (after meningitis), and suffered from giddiness and tinnitus, especially on the left side. The tympanic membranes were normal, but the patient was quite deaf: there was horizontal nystagmus on looking to right or left, but none on syringing the ears with hot or cold lotions, or on turning ten times to right or left. On testing the galvanic excitability of the vestibular part of the eighth nerve, it was found that, with the kathode at the right ear, a current of ten milliamperes produced rotatory nystagmus to the right when the patient looked straight forward. The anode at the right ear, with a current of twelve milliamperes, produced nystagmus to the left. In the left ear the kathode, with a current of twelve milliamperes, produced nystagmus to the right, and the anode, with only six milliamperes, nystagmus to the left. The results of the goniometer test are also given—the difference between the results with “eyes open” and “eyes closed” is very striking.

The second case is similar to the first. Dr Mackenzie comes to the following conclusions:—

1. Subjective noises are increased on one side by applying the kathode to the ear—on the opposite side when the anode is in contact with the ear.
2. Increase of the subjective noises of the right ear occurred with the same strength of current when the kathode was applied to the right ear, or when the anode was in contact with the left ear.
3. With the current of four milliamperes kathodal closure gives an increase of the subjective noises on the same side, and at the same time the noises in the opposite ear disappear.
4. With a current strength of four milliamperes kathodal opening increases the subjective noises only in the opposite ear.
5. Anodal closure affects the opposite ear, and anodal opening the ear on the same side.

From these observations Dr Mackenzie attempts to draw some conclusions as to the value of operation (section) on the auditory nerve in cases of severe tinnitus; she also thinks that in the two cases investigated the pathological process had advanced further

in the vestibular than in the cochlear nerve, and that this is the reason for the cochlear nerve reacting to weaker currents: if this be so, it is very unusual, as the cochlear nerve is the one which shows most marked degeneration in such cases when examined microscopically.

J. S. FRASER.

DIAGNOSIS AND TREATMENT OF NEURASTHENIA. JOHN K. (271) MITCHELL, *Johns Hop. Hosp. Bull.*, Vol. xix., No. 203, Feb. 1908.

NEURASTHENIA is defined as a chronic fatigue, due in part to malnutrition, in part to functional over-exertion, occurring in persons with a predisposition hereditary or acquired.

Its diagnosis is said to rest on certain subjective symptoms, which fall into four main groups:

(1) Fatigue symptoms: general weariness, lassitude constantly present, greatly increased by the smallest exertion.

(2) Irritable weakness symptoms: increased reflexes, cardiovascular disturbances, muscular tremors, excessive emotionality.

(3) Disorders of will, such as lack of self-control, indecision. Sir James Paget stated this cardinal symptom epigrammatically: "The patient says he cannot; the nurse says he will not; the fact is, he cannot will."

(4) Psychic depression, and anxiety or fear symptoms. Sometimes there is simple depression, sometimes definite fears or "phobias."

Negatively there is a fifth very important symptom—namely, the absence of any typical or peculiar organic changes.

In regard to treatment, the first requirements are met by rest, or rather by rest treatment, and the latter by a rational system of education of the will, the logical faculty, and the reasoning powers of the patient. The amount of rest should be regulated by the special needs of the case, varying from a couple of hours' quiet to long-continued complete recumbency. In slighter cases isolation is not necessary.

D. K. HENDERSON.

PSYCHASTHENIA. JOSEPH COLLINS, *N.Y. Med. Jour.*, Vol. lxxxvii., (272) No. 7, Feb. 1908.

JANET suggested psychasthenia as the name for a disorder characterised by mental, emotional, and physical symptoms, made up principally of obsessions or imperative concepts, fears, doubts, anguish, uncontrollable movements, enfeebled will power, and some or all of the customary physical symptoms of neurasthenia. The patient can realise how strange and morbid the obsession is

and can discuss it in an impersonal way, without it affecting the tenaciousness of the obsession.

The most complete and typical case the writer has ever seen is fully recorded.

D. K. HENDERSON.

MORBID SOMNOLENCE. D'ORSAY HECHT, *Am. Journ. Med. Sc.*, (273) Vol. cxxxv., No. 3, March 1908.

THE clinical report of a case of narcolepsy, occurring in a man aged 48 years, who had contracted syphilis when 18 years of age.

The case was first seen in 1906, the sleeping having gradually increased in frequency and duration since 1895. Little attention was paid to the "naps" until one day the patient fell asleep while making a bed, and on another occasion "suddenly dropped off" in the course of a conversation. He experiences an unconquerable desire to doze about every three hours, and, if not disturbed, sleep lasts from one half to one hour. The sleep is perfectly refreshing, and so light that he is wakened by the slightest touch, and nothing said within hearing distance escapes him. Keeping constantly on the move, or doing real, active, manual labour is his only hope of keeping awake.

Numerous writers on the subject are quoted.

D. K. HENDERSON.

PATHOGENY AND TREATMENT OF SEA-SICKNESS. (Le mal (274) de mer. Essai de pathogénie et de traitement.) L. MAILLET, *Thèses de Paris*, 1907-8, No. 142.

ADOPTING the views of F. Regnault, Maillet distinguishes between a psychic and a somatic sea-sickness. The former is due to suggestion alone, and is curable by suggestion. Psychic naupathy may be recognised by the patient complaining of symptoms which are never found in somatic naupathy, *e.g.*, diplopia or anuria, or by the symptoms occurring in fine weather or on terra firma.

Psychic naupathy is essentially contagious, and is most likely to occur in nervous persons who are subject to other forms of locomotor vertigo, *e.g.*, carriage or railway sickness. It may also be found in animals, which, though also subject to somatic sea-sickness, are more liable to suggestion than most men.

In the treatment suggestion may be employed either directly or indirectly, *e.g.*, by drugs. In such cases the quality of the remedy is of less importance than the assurance with which it is prescribed. The success that has followed a countless number of "specifics" Maillet attributes to their action on a purely psychic naupathy. Treatment by auto-suggestion is very efficacious.

Numerous observers have noted that the symptoms have become attenuated, or have entirely disappeared, by the vigorous exercise of will power.

Somatic naupathy has received various explanations. The most probable is the abdominal theory proposed by Kéraudren. The swaying of the vessel produces movements of the abdominal viscera which irritate the semilunar ganglia. The symptoms are thus produced by a reflex path. It must be noted that numerous cases occur of combined psychic and somatic naupathy. The treatment of somatic naupathy consists in the administration of sedatives, *e.g.*, opium, morphia, or cocaine, in the constriction of the abdomen by a belt, and by immobilisation in the recumbent position.

J. D. ROLLESTON.

THE SYMPTOMS DUE TO CERVICAL RIBS. WM. THORBURN,
(275) *Med. Chron.*, Dec. 1907, p. 165.

NOTES of a number of cases are given in this article, the nervous symptoms being considered in greatest detail. It would appear that symptoms are much more common in the female than in the male, that they generally involve the right upper limb, and that they tend to appear in early middle life. The cases in which nervous symptoms are present fall into two groups—the neuralgic, in which pain and subjective weakness are alone complained of, and the paralytic, in which there is definite loss of power or sensation. In the neuralgic cases the pain may be felt in the neck or in the upper limb, but is much more characteristic in the latter. It follows the distribution of the first dorsal, or first dorsal and eighth cervical roots; it is usually described as tingling, or as a numb pain, and is often associated with a marked feeling of coldness, and is apt to be aggravated by cold.

The more serious paralytic cases commence with purely subjective symptoms; when objective symptoms are found, they also are usually limited to the regions supplied by the first dorsal and eighth cervical roots, *e.g.*, paralysis and wasting of the hand muscles and sensory defects, frequently with dissociation of the various forms of sensation. Early removal of the offending rib is recommended in all cases of reasonable severity. Details of the operation are given, the author giving preference to a straight, more or less vertical incision in the posterior triangle, reaching well up above the clavicle and lying well back, so that in the necessary dissection the nerves and subclavian artery are turned forward—the operation being from the side rather than from the front of the neck.

J. H. HARVEY PIRIE.

**THE RELATION BETWEEN SCIATICA AND DISEASE OF THE
(276) HIP-JOINT.** W. IRONSIDE BRUCE, *The Practitioner*, April
1908.

DR WILLIAM BRUCE five years ago thoroughly discussed the clinical aspect of the theory that a relation existed between sciatica and disease of the hip-joint.

The author of the present paper seeks to offer, by means of radiograms of the hip-joint in cases of sciatica additional proof of Dr William Bruce's contention—namely, that the origin of pain in sciatica is in the hip-joint, the sciatica referred to being understood to exclude examples of sciatica due to manifest spinal disease or to pressure from tumours in the pelvis.

The possibility of recognising, by means of the X-rays, changes in the normal appearance of joints affected by gout or rheumatism is demonstrated by means of radiograms.

There are quoted twelve cases presenting typical symptoms of sciatica in which an associated arthritis of the corresponding hip-joint was discovered. Five of these are illustrated by radiograms.

A few of the clinical arguments in support of Dr William Bruce's contention are advanced; but the main object of the paper is to bring under notice five cases which presented the signs and symptoms usually recognised as sciatica, in all of which this diagnosis had been made independently, and the usual treatment for the condition been carried out. In each of them the radiogram shows disease of the hip-joint more or less extensive in its nature.

The conclusion is, that if systematic examination of the hip-joint by X-rays, in intractable cases of sciatica, be carried out, it will be found in many of the cases that the sciatica is present only as a symptom of disease of the hip-joint. AUTHOR'S ABSTRACT.

PSYCHIATRY.

INSANITY, SIMULATION, AND CRIMINALITY. (Locura, Simu-
(277) lacion y Criminalidad.) JOSE INGENIEROS, *Arch. de Psig. y
Crim.*, Jan.-Feb. 1908.

IN an article of some length Prof. Ingegneros publishes an account of a delinquent whom he describes as one of the most complex and interesting cases, and one which has evoked the most divergent expert opinions since the foundation of the Institute of Criminology at Buenos Aires. The man, Alexandro Puglia, was a Neapolitan, æt. 38, with a marked neuropathic heredity, who at the age of twenty-eight emigrated to South America. Official documents obtained from Italy showed that he was of an impul-

sive, violent, and anti-social character, of bad education and surroundings, a Camorrist, potator and syphilitic, and that he had been in prison in Rome in 1895, whence he was sent to the asylum of St Maria for mental observation. There he presented auditory hallucinations and persecutory mania of brief duration. Later in the same year, whilst undergoing military service, he was again under observation in the asylum at Florence, on account of his violent and refractory behaviour, and was diagnosed as a degenerate, subject to episodic psychoses, but not truly insane, and discharged as unfit for the army. In Buenos Aires he was thrice imprisoned for fraud, in 1898, 1899, and 1901, and again in 1905 for homicide. Whilst awaiting trial on the last charge, he made an attempt (?) at suicide, was passed to the infirmary, where he simulated insanity. At the infirmary he was carefully examined, was found to present numerous physical stigmata of degeneration, had unequal and sluggish pupils, tongue tremor, slight articulative difficulty and auditory hallucinations, and delusions of persecution, which were considered to be simulated, as they were at variance with his conduct. The medical officer of the infirmary therefore advised the Court that the prisoner was not insane, and was a malingerer who was at the time of their observation in a normal mental condition. The prisoner was therefore sentenced to ten years' penal servitude. He again simulated insanity, and his sentence was increased to fifteen years by the Chamber. Shortly before entering upon this sentence he stabbed a fellow-labourer, and the question of his mental state was reopened. In reviewing the whole facts, Prof. Ingegnieros comes to the following conclusions:—

1. The prisoner is a mental degenerate, with intercurrent episodic attacks of excitement, with a profound lack of moral sense, aggravated by chronic alcoholism.
2. His simulation was the simulation of an insane person—*i.e.* was supra-simulation.
3. He should be considered irresponsible with regard to his last homicidal assault.
4. As his mental derangement is a permanent condition, he ought to be permanently sequestered as a criminal lunatic.

Those who are intimately acquainted with convict establishments will be able to recall many cases like this of Prof. Ingegnieros, cases in which prisoners of degenerate physical type, intellectually of average parts, but cunning, suspicious, inordinately egotistical, refractory, and liable to periodic explosive attacks of excitement and violence, during which apparently genuine auditory hallucinations and delusions of persecution develop on an original groundwork of morbid suspicion, have

been futilely punished over and over again for offences committed in and outside of prison, and have finally been certified as insane. The simulation of insanity in such cases is not uncommon, so much so, indeed, that the simulation of insanity is itself strongly suggestive of mental defect. As Lasèque said—" *On ne simule bien que ce que l'on a.*"

R. CUNYNGHAM BROWN.

PARALYSIS AGITANS IN AN INSANE EPILEPTIC. (*Maladie (278) de Parkinson survenue chez une démente épileptique.*) H. BOURILHET, *Gaz. des Hôp.*, jan. 7, 1908.

THE author describes the following clinical case:—The patient is seventy-six years of age; she has suffered since the age of twenty-seven from recurring mania, characterised by attacks of hallucinatory delirium, followed by vertigo. In the interval between the attacks the patient is calm, but sometimes dangerous and impulsive. Towards the age of fifty there appeared attacks of epilepsy, which increased in frequency after the menopause. Then her intellectual faculties diminished, and at the present time she is insane. She now presents the appearance of a case of paralysis agitans with the characteristic mask, attitude, propulsion, and tremor. The case is thus one of paralysis agitans occurring in an insane epileptic.

The author mentions a similar case reported by Combernale, but he refrains from putting forward any theory, and even from establishing any causal relation between the symptoms of paralysis agitans and those of the epilepsy.

O. CROUZON.

ADIPOSIS DOLOROSA IN A CASE OF DEMENTIA PRÆCOX. (279) FURSAC et PASCAL, *L'Encéphale*, Feb. 1908, p. 131.

THE case is that of a woman of 65 who has been in an asylum with dementia præcox since 1882. The adiposity had developed subsequently to her admission, but its development had not been traced. The disposition of the masses of fat is characteristic, the face, front of neck, hands, and feet escaping. Spontaneous pain appears not to be present, but it is readily evolved by pressure on, or movement of the skin over the masses of fat. The thyroid is not palpable. The authors attribute the common association of Dercum's disease with mental troubles to the action of some still unknown intoxication on an organism with a neuro-psychopathic heredity.

J. H. HARVEY PIRIE.

- HOW LUNATIC ASYLUMS SHOULD BE CONSTRUCTED.** (Como (280) *deben ser los Asilos para Alienados.*) A. GALCERÁN GRANÉS (Med. Supt. Reus Asylum, Spain), *Arch. de Psiq.*, Jan.-Feb. 1908.

THE author gives an interesting account of the development of asylums in Spain, their actual state to-day, and the direction in which he considers they would be improved by the establishment of sanatoria for psycho-neurotics; special establishments for epileptics; such alterations in the structure of asylums as would permit of better classification; the extension of the "open door" and "no restraint" to all suitable cases, and the provision of reformatories for moral degenerates.

R. CUNYNGHAM BROWN.

- PSYCHIATRIC EXPERT EVIDENCE IN CRIMINAL PROCEED-**
(281) **INGS: ITS IMPERFECTIONS AND REMEDIES.** G. W. JACOBY (of New York), *N.Y. Med. Journ.*, March 7, 1908.

THE present methods of securing, maintaining and remunerating expert evidence are discussed and frankly condemned. The plan of permitting experts to be retained "For the defence" and "For the prosecution"; the payment of extraordinary fees in competition; the chances of the defence when invalidated by poverty are all commented upon most pertinently.

The author proposes the establishment by the State of a permanent institution of medical experts selected according to fitness, and uninfluenced by politics. An age limit, special training and special examination should be considerations upon which a "Physician to the Court" should be selected. His remuneration should be apportioned in accordance with the difficulties and length of the case.

The laws of evidence are criticised and the "obstacle race" denounced. The expert should finish his exposition in continuity, frankly and honestly founded upon the facts of the case; questions from the defence, prosecution, court and jury respectively should then be allowed. Much advantage would be realised if the defendant suspected of mental disease was committed for continued observation to some regular State hospital, and a written report submitted by the resident experts.

Finally, the author refers to the absurdity of adhering to the "right and wrong" tests of the penal code handed down from English law, and points to Germany as the example of a country where regulations such as he proposes are in vogue.

C. H. HOLMES.

TREATMENT.

ARTHRODESIS AND TENDON TRANSPLANTATION IN PARALYTIC CONDITIONS. ROBERT JONES, *Brit. Med. Journ.*, March 28, 1908, p. 728.

A VALUABLE address on the above subject was delivered by Mr Robert Jones, at the Medical Graduates College and Polyclinic. The following are some of the more important points insisted on by the author. The joints most amenable to treatment by arthrodesis are the ankle, the mid-tarsal, and next to these the knee. The operation should not be performed before the eighth year. The author's earlier failures were mostly due to non-observance of this rule. Such early operations are apt to result in feeble fibrous ankylosis, and later on, from the sacrifice of too much cartilage, in irregularity of growth. This irregularity of growth, in the case of the ankle, usually produces a deformity of the pure varus type. To avoid this, the foot should be placed in an over-corrected position, a wedge of bone being taken if necessary from the astragalus, with its base to the outer side. If the wedge is taken from the astragalus the growth of the limb is not interfered with. Arthrodesis is also contra-indicated where the surgeon is not satisfied that the muscles are paralysed beyond hope. At least two years must have elapsed in the case of muscles suspected to be completely paralysed. In many cases such muscles recover partially or completely in a surprising manner if proper measures are taken. Although wasted, and not responding to electricity, these muscles are not really paralysed but *over-stretched*, and restoration of function may occur in a few months if structural shortening is allowed to take place in their bodies. This is brought about by excising a portion of their tendons with immediate resuture, supplemented if necessary by division and elongation of their healthy but tense opponents, and sometimes, in addition, by the transplantation of healthy tendons. The joint is then kept at absolute rest in a splint in the corrected position, and electricity and massage diligently and persistently applied for a considerable period. The keynote to treatment, however, is to relieve such over-stretched muscles from all strain. In many cases also Jones finds the removal of an elliptical portion of skin over the joint of the utmost value. When the cut edges are united, if enough has been excised, it will be found that the joint is kept in the proper position. The skin excision, in fact, acts as a splint, and maintains the over-correction. Cases so treated may never require arthrodesis, and, if such should eventually become necessary, the conditions are much more favourable for its performance than before. Instances have occurred in drop wrist where the over-stretched extensors

have regained their contractility after seventeen and nineteen years (Tubby and Jones).

The removal of skin flaps from the proper aspect of the joint is also very valuable in arthrodesis and in tendon transplantation. With regard to the latter operation, Jones usually waits till the child is five years of age. The sheath of the transplanted tendon should be spared as much as possible, and should accompany it to its destination. He has given up transplanting into tendons; the tendon should be sutured to the periosteum, or into a tunnel in the bone. The suture fixing it should be tight. Silver has shown that it is practically impossible to tie and suture tightly enough to cause necrosis. The tendon must pass straight to its destination, and not in an angular manner. To avoid such deflection, it is often necessary to tunnel the soft tissues. The transplanted tendon must not be subjected to undue tension, or it will wither.

The patient, in all these operations, must not be allowed to use the limb fully for a very considerable time, otherwise relapses will occur.

A. A. SCOT SKIRVING.

ORTHOPÆDIC THERAPY DURING THE EARLY STAGES OF
(283) **ACUTE ANTERIOR POLIOMYELITIS.** NUTT, *N.Y. Med. Journ.*, 29th Feb. 1908.

PROPER and painstaking treatment, instituted during the acute stage, will greatly lessen the extent of the crippling results of this disease, and be the means, in some cases, of a complete cure. The affected parts must be kept in as healthy a condition as possible by massage, electricity, and hydrotherapy. Scientifically applied massage, as contrasted with mere rubbing, should be performed daily for from ten minutes to half an hour. Each muscle should be treated by the electric current for three or five minutes daily. The faradic current is used when any response to it is obtained, otherwise the constant current is employed. The local temperature is to be maintained by extra clothing, constantly worn; hot water bags and electric heaters only have a temporary effect on the lowered temperature, and are not recommended. Passive movements, devised to preserve the normal range of action of the joints, must be commenced at the beginning and carried out daily; otherwise muscle-shortening, which sets in very quickly, may render normal movement without force impossible within six weeks. The over-stretching of paralyzed and weakened tissues must be prevented by the constant wearing of suitable apparatus, which counteract unparalyzed antagonistic structures and the effects of gravity. Even after years of neglect, overstretched muscles may partially recover their normal functions as a result of suitable

treatment. Prolonged rest in bed is undesirable. Careful consideration must be given to each individual case with reference to the selection of suitable restraining apparatus, and the confined joints and muscles should be exercised twice daily by passive and resisted active movements performed with the apparatus removed.

HENRY J. DUNBAR.

ASYLUM TREATMENT FOR INEBRIATES. (*Asilo para Bebedores*.) PEDRO DORADO (Univ. Salamanca), *Arch. de Psiq.*, Jan.-Feb. 1908.

PROF. DORADO, in the lengthy conclusion of a previously published article, reviews the statistical information bearing upon the relation of alcoholism and crime; the parallel fluctuations of alcoholic consumption and numbers of delinquencies; the various legal measures which are in force in all countries which legislate on this matter, either by penal measures, interdiction, control in State establishments, or control for the purpose of reformation, and finally examines the results of treatment in inebriate homes. The author accepts without demur the statistical evidence in favour of alcohol as an enormously important cause of criminality, and pleads for the legal enforcement of asylum treatment for all drunkards, whether they have committed breaches of the law or not, the treatment to be curative and not punitive in aim. The author is evidently not in agreement with the Spanish Minister of the Interior (quoted by Dr Branthwaite, the English Inspector under the Inebriates Acts Report for 1901), who said: "Fortunately the vice in question does not constitute a social peril in Spain."

R. CUNYNGHAM BROWN.

Reviews

ELEMENTS OF PSYCHOLOGY. SYDNEY HERBERT MELLONE, D.Sc., and MARGARET DRUMMOND, M.A. Edinburgh: Blackwood, 1907, pp. xvi, 483. Price, 6s.

THE text-books on psychology recently published in this country and America offer a perplexing problem to the student, both in their number and in their variety of treatment. The present volume is intended primarily for the beginner; it is mainly conservative in its standpoint; the experimental and biological aspects of psychology receive, it may be thought, inadequate considera-

tion ; and the traditional order of subjects is inverted, the authors proceeding from the complex to the simple, from Mental Activity, Conation, Attention to Feeling and Emotion, from these to Sensation, Perception, Memory, and Conception.

There can be no doubt that for the beginner the work admirably fulfils its purpose ; it is written with an attractive simplicity of style ; the illustrations are drawn in many cases fresh from the author's own experiences, in others from the best experimental work that has been done ; the more difficult questions are referred to in sections of smaller type than the main subjects ; there are many useful quotations from the more systematic works, and ample references to the literature of psychology. Most teachers will agree that Sensation and Perception are the hardest parts of the subject for the average student ; the attention given to them at the beginning of a course is also apt to distort his view of their importance relatively to Feeling and to Mental Activity. On the other hand, no discussion of the latter is possible without referring to certain classes of sensations—the organic and the kinæsthetic—which, in fact, are dealt with by the authors in the chapters on Feeling and Mental Activity, and therefore *before* the special sensations. This seems neither a logical nor a practical order of study. The real difficulties of Sensation and Perception must be overcome rather by a more extended use of experiment. As Wundt long ago argued, experiment in psychology does not merely aid or control observation, it offers the only conditions under which observation is possible. A mind trained to observe the characters and changes of sensation will more readily appreciate the subtle influences at play in Emotion and in Volition.

The more advanced student also will find much that is of value to him in this manual ; the greater questions of psychology are treated broadly, impartially (with rare exceptions, perhaps), and always with full references to the sources. The portions on Mental Activity, on the Psychology of Learning (pp. 158 ff.), on the psychological theory of Pleasure and Pain (250 ff.), and on Memory (ch. xiii.) may be particularly referred to. Three marked features of this more detailed work are: the application of the "hierarchical conception" of the nervous centres (Houghlings Jackson) to mental life (pp. 32, 71, 113, etc.), the adoption of the interaction theory of the relation of mind to body, and the abundant use of the idea of "psychological dispositions" or tendencies, as real factors, in the explanation of memory, habit, will, sentiment, etc. It is doubtful whether the last two principles are consistent with each other, and still more doubtful whether the argument adopted in defence of interaction will hold, viz., that of a distinction between the *direction* and the *quantity* of physical energy. It is, however, a regrettable necessity that even elementary text-

books in psychology must still contain polemical matter. It has its uses, of course.

There can be little hesitation in placing this book among the best of its kind.

J. L. M'INTYRE.

DIE GESCHWÜLSTE DES NERVENSYSTEMS. LUDWIG BRUNS.
2te Auflage. Berlin: S. Karger, 1908.

THIS work is a standard one, and its second edition, appearing ten years after the original, will be read with keen interest. The book consists of three main divisions. The first of these deals with brain tumours, the second with tumours of the vertebral column and spinal cord, whilst the third is devoted to tumours of the cranial and spinal nerves in their extra-cranial and extra-vertebral course, and to paraneural neoplasms. Each of these subjects is treated with the thoroughness and completeness which are to be expected from the distinction of the author. In a short review it is impossible to give more than a general idea of the wealth of clinical observation here contained; every page is worthy of study, whilst the clearness of thought and of diction is particularly commendable, even in the most obscure and difficult divisions of the subject. Another point which cannot fail to strike the reader is the author's candour when he deals with doubtful or disputed points, and the scrupulous fairness with which he states opposing views, including those contrary to his own.

Under the first heading, that of brain tumours, we find a chapter devoted to the pathological anatomy of the different intra-cranial growths, including not only neoplasms proper, but infective granulomata, parasitic cysts, and aneurisms. The etiology of brain-tumours is then discussed. With regard to the curious predilection of tuberculous growths for the posterior fossa, Bruns offers the suggestion that this may be associated with the close connection which subsists between the deep cervical lymphatic glands and the posterior parts of the brain. As regards the influence of trauma upon the subsequent appearance of a brain tumour, Bruns is of opinion that in most cases the tumour is pre-existent to the injury, and that trauma does not produce the tumour, but simply renders it more manifest. He admits, however, that in cases of tuberculous and syphilitic growths, trauma may conceivably determine the locality of the mass, though it cannot be the prime cause.

There is an interesting discussion of the local effects of a tumour upon adjacent and remote regions of the encephalon. The symptoms of increased intra-cranial pressure he attributes, with emphasis, to the accumulation and increased tension of the cerebral fluid. To this increased fluid pressure he also attributes

the main etiological factor in the production of one of the most important clinical symptoms, choked disc. Bruns is a strong supporter of the mechanical origin of optic neuritis, rejecting the inflammatory and toxic theories.

The general symptomatology of brain tumours is then dealt with. He distinguishes between focal symptoms proper, symptoms of implication of neighbouring parts, and symptoms of affection of distant parts. It is interesting to note that he denies any special preponderance of psychical symptoms in frontal growths, in this respect differing from the opinion of Beevor and most British neurologists. Amongst clinical methods of examination, he calls attention to the value of percussion of the skull, noting the degree of local tenderness, and also the presence or otherwise of a peculiar percussion-note, sometimes a crack-pot sound resembling that of a pulmonary cavity. Such percussion changes, however, are of value only when they coincide with other clinical localising phenomena. By themselves they are insufficient for focal diagnosis. The crack-pot note, in particular, is not pathognomonic of tumour, but merely indicates a local thinning of the cranial bones.

Then follows a most complete and accurate account of the focal symptoms of tumours in the different parts of the encephalon. Space forbids us to enter into details; the account is uniformly excellent.

The course and prognosis of intra-cranial growths are then discussed. Attention is drawn to the occasional, though rare, cases of spontaneous improvement, and even of cure, as by calcification of tuberculous or other growths, coagulation of aneurisms, etc. Bruns agrees with other observers in noting that gummata are sometimes resistant to anti-specific treatment, and that certain cases of tuberculous and even of sarcomatous growths may be temporarily benefited by mercury and iodides. The list of conditions from which intracranial growths have to be distinguished is a long one; it includes plumbism, anæmia, arterio-sclerosis, migraine, empyema of accessory nasal sinuses, Menière's syndrome, brain abscess, purulent and tuberculous meningitis, gummatous disease of the meninges, dural hæmatoma, cortical encephalitis, cerebral hæmorrhage, thrombosis and embolism, acquired hydrocephalus, acute serous meningitis, multiple sclerosis, paralytic dementia, epilepsy, trigeminal neuralgia, uræmia, hysteria, neurasthenia, and hypochondriasis. All of these are carefully discussed. The value of skiagraphy in certain cases is emphasised, and it is pointed out that shadows may be cast not only in tumours, which widen or deform the sella turcica, and by calcifying growths, but occasionally by hard fibromata and other tumours. Bruns approves, in certain cases, of Neisser and Pollack's exploratory puncture of the skull and brain with aspiration of a fragment of

tumour. The conditions under which such puncture is justifiable are, however, somewhat inconclusively stated. For example, he suggests this procedure in cases where the diagnosis lies between a frontal and a cerebellar growth, or where the physician is in doubt as to which symptoms are focal and which the result of pressure on adjacent parts. Many neurologists will be inclined to disagree with this point of view.

The diagnosis of the nature of the growth is then discussed. The importance of attempting a pathological diagnosis is emphasised, in view of the fact that some growths (for example, sarcoma, endothelioma, fibroma and neuro-fibroma, tubercle, cysticercus and echinococcus cyst) are generally sharply marked off from the neighbouring brain tissue by a zone of softening, whilst glioma and carcinoma are truly infiltrating tumours, and have, therefore, much less favourable chances of removal by operation. Accurate pathological diagnosis is commoner in the parasitic cysts, especially cysticercus, in aneurism and gumma and in certain cases of neuro-fibroma, and of course in metastatic carcinoma and sarcoma, whilst it is difficult and often impossible in primary sarcoma, endothelioma, fibroma, cholesteatoma, and psammoma. Tuberculous growths, in this respect, occupy a middle position.

In the diagnosis between a cortical and a sub-cortical growth Bruns lays special emphasis on the value of local percussion of the skull. The presence of tenderness and local alteration of percussion-note he regards as strong corroborative indications of a local cortical growth.

As to the surgical treatment of brain tumours, Bruns's statistics lead him to the conclusion that only some 35 per cent. of intra-cranial growths are surgically removable. This does not mean that only 35 per cent. are to be operated on, for palliative operation should be recommended in many cases where enucleation of the growth is impossible, such palliative operation being for the purpose of relieving headache and of preventing blindness by the relief of optic neuritis. This part of the book concludes with an account of the methods of cranio-cerebral topography and of electrical exploration of the cortex.

The second division of the book deals with tumours of the vertebræ and of the spinal cord and its meninges. In view of the relative frequency with which spinal caries produces secondary changes in the cord, one could wish that this subject had been dealt with as exhaustively as that of the neoplasms proper. Still Bruns draws attention to the main points of resemblance and of difference between vertebral growths and spinal caries. Curiously enough he makes the statement that in caries, as contrasted with neoplasms, herpes zoster has not yet been observed as a symptom. This is not accurate, for I have seen at least one case of typical

zoster from tuberculous disease in the neighbourhood of the corresponding spinal ganglion. In the diagnosis between tumours of the vertebral column and those of the spinal meninges, Bruns states the useful dictum that in vertebral tumours the sequence of symptoms is first bony pain and deformity, secondly root-symptoms, and thirdly symptoms of spinal cord affection, whereas in extra-medullary growths the sequence is firstly root-symptoms, then spinal-cord phenomena, and bone symptoms last, if at all.

The difficulty of diagnosis between intra-medullary and extra-medullary growths is admitted. The chief points of value in this connection are that in intra-medullary cases root-symptoms and symptoms of interruption of spinal tracts occur practically concurrently and not successively one after the other, the stage of pure root-symptoms being generally absent; further, in central gliosis the symptoms are of syringomyelic type, whilst in tumour or sarcoma of the anterior cornu the phenomena are those of a rapidly progressive muscular atrophy of spinal type. Intra-medullary growths tend to produce bilateral symptoms from the outset, instead of commencing unilaterally as in most extra-medullary growths.

As to the prognosis of spinal-cord tumours, Bruns points out that in spite of their infrequency (occurring only one-sixth as often as brain-tumours), they are relatively much more amenable to surgical treatment, and therefore once the diagnosis is established (gummata being excluded), operation should be urged. The older the patient, the more likely is the growth to be capable of complete removal.

The third and last division of the book deals with tumours of peripheral nerves—neuromata, and of structures in their neighbourhood—para-neural tumours. The true and false neuromata, with all the subdivisions of the latter, are well described, together with the tuberculous, gummatous, and leprous affections of the peripheral nerves.

The book is sure of a wide circulation amongst clinicians; its possession, in fact, is well-nigh indispensable to the neurologist.

PURVES STEWART.

STUDIEN ÜBER POLIOMYELITIS ACUTA. (Zugleich ein Beitrag zur Kenntnis der Myelitis acuta.) IVAR WICKMAN. Berlin: Karger, 1905, M. 6.

BEITRÄGE ZUR KENNNTNIS DER HEINE-MEDINSCHEN KRANKHEIT. (Poliomyelitis acuta und verwandter Erkrankungen.) IVAR WICKMAN. Berlin: Karger, 1907, M. 6.

THESE two volumes form an important contribution to our knowledge of acute poliomyelitis and allied diseases. The

author has combined the names of Heine, who was the first to show the existence of a lesion of the spinal cord, and of Medin, who first described the epidemic form of the disease, to include the different forms which were found to occur during the extensive epidemics in Sweden in 1905.

The first-named work gives an account of a series of cases of acute poliomyelitis, and of the microscopic and macroscopic appearances found in them. It is illustrated by beautiful microphotographs of the pathological changes present, and the writer summarises his views as to the nature of the disease in a number of conclusions, of which the following are the most important:—

(1) Acute poliomyelitis is a disseminated infiltrative myelitis, associated with oedema and interstitial changes, which may be limited to the neighbourhood of individual ganglion cells, or to groups of these, being as a rule most marked in the cervical and lumbar enlargements.

(2) The changes are not limited to the anterior horns, but may extend to the rest of the grey matter in the lumbar and lower dorsal regions, affecting the columns of Clarke as severely as the anterior horns, or even more so.

(3) The changes may also extend to the white matter and to the pia mater.

(4) The cellular infiltrations are related to all the vessels, arteries, and veins, whether central or peripheral, and show no preponderating dependence on the central artery.

(5) There is no evidence of an embolic origin of the disease.

(6) The interstitial and parenchymatous changes have an approximately parallel course, and there is some evidence that the pathological changes depend more upon a lymphatic than upon a vascular spread.

(7) No bacteria have been shown to be the cause of the disease.

(8) The changes in acute adult poliomyelitis are entirely similar to those in the infantile spinal paralyses, and the main cause of the destruction of the nervous elements must be looked for in the interstitial inflammation.

The second volume is dedicated as a *Festschrift* to Professor Medin, of Stockholm, on his sixtieth birthday, in honour of his being the first, in 1890, to bring before the profession, at the International Medical Congress in Berlin, the epidemic form of the disease, to show that poliomyelitis is intimately related to other processes in the medulla, pons, and cerebrum, and to prove the teaching of Strümpell and Marie as to the infective nature of the disease. In it Wickman classifies the cases according to various forms:—(1) Poliomyelitic form; (2) Form of Landry's paralysis; (3) Bulbar or pontine form; (4) Encephalitic form;

(5) Ataxic form; (6) Polyneuritic form; (7) Meningitic form; (8) Abortive forms. The symptomatology of each of these is fully described, and the writer gives a valuable discussion of the relationship of the polyneuritic to the poliomyelitic form, as well as of their relationship to the meningitic variety. He goes into the question of the Swedish epidemic of 1905 in great detail, showing that it was most prevalent from July to October; that it spread all over Sweden from north to south in an irregular manner, following lines of communication, and involving the country districts more than the towns (72 town cases as against 959 country ones); that it tended to occur in foci, and to be carried by the intermediation of healthy persons, themselves unaffected, as well as by direct contact, and that it might affect several persons in the same family or house, lingering in a neighbourhood from one to several months.

The author states that prognosis as to life is unfavourable to a degree that has not hitherto been believed, and that in older children and in adults it is much more grave than in younger children, but that prognosis as to complete recovery is, on the other hand, much less serious than is generally accepted, in so far as numerous cases (those included under "abortive forms") have no paralysis, and that not a few in which paralysis has occurred completely recover.

ALEXANDER BRUCE.

**PATHOLOGISCH-ANATOMISCHE UNTERSUCHUNGEN ÜBER
AKUTE POLIOMYELITIS UND VERWANDTE KRANK-
HEITEN VON DEN EPIDEMIEN IN NORWEGEN, 1903-
1906. FRANCIS HARBITZ und OLAF SCHEEL. Christiania:
Dybwad, 1907.**

THE authors describe a number of cases of acute poliomyelitis which came under their observation during the epidemics in Norway, and discuss their pathological conditions, as well as the etiology and pathogeny of acute myelitis.

This valuable work is illustrated by a series of coloured plates, which show very beautifully that the incidence of the disease is not limited to the anterior cornua, but that the inflammation may affect the posterior cornua and the white matter of all the columns, as well as the pia mater, and that it is not limited to the spinal cord, changes of vascular congestion and round-celled exudation being found in the whole of the central nervous axis. The work materially supports that of Wickman, and broadens our knowledge of the distribution, etiology, and nature of acute poliomyelitis.

ALEXANDER BRUCE.

DIE FUNKTIONEN DER NERVENCENTRA. Prof. Dr W. v. BECHTEREW. Deutsche Ausgabe von Dr RICHARD WEINBERG. Erstes Heft. Jena: Gustav Fischer, 1908.

THIS volume, which extends to almost 700 pages, is the work of a veteran neurologist who has already enriched the literature of his subject by several standard works. It is a German edition issued by Dr Weinberg in conjunction with the author, and as such is accessible to a larger circle of readers than it would be if published in the Russian language.

The first 82 pages are taken up with a description of the different experimental and clinical methods that have been employed in investigating the functions of the central nervous system, and with a general review of the physiology of nerve conduction, reflex action, inhibition, and co-ordination. The relationships between afferent (sensory) and efferent (motor) impulses are considered, and the importance of the state of the posterior roots of the spinal nerves in all limb and body movements is emphasised.

The rest of the volume is devoted to a consideration of the various nerve centres in the spinal cord and medulla oblongata, and of the nervous mechanisms by which these govern and control the functions of the different organs of the body. These are given in great detail, and the descriptions are illustrated by many original schemata. The last chapter is taken up with the conducting paths in the spinal cord.

The value of the book is increased by the fact that in it is embodied much original work by the author and his pupils, which has not been published elsewhere. It contains a very full bibliography, and as a work of reference it will be of great value. The recent teaching of Sherrington on reciprocal innervation does not appear to be duly appreciated.

SUTHERLAND SIMPSON.

LES TRAITEMENTS DU GOITRE EXOPHTALMIQUE. PAUL SAINTON et LOUIS DELHERME. Paris: J. B. Baillière et fils, 1908. Pp. 96. Price, 1.50 fr.

As Professor G. Ballet says in his Preface, "il n'y a pas *un* traitement, mais *des* traitements." Each case must be treated on its own merits. A choice of methods must be made, and this little volume, where they are all passed in review, and their value criticised, and the indications and contra-indications given, will give considerable help in this often difficult task. The authors recommend serum of dethyroidised animals in large doses for typical cases. Electricity, particularly galvano-faradisation, should be regularly tried. Among drugs they give sodium salicylate the

first place, and quinine the next, but varied symptomatic treatment is often required. Surgical treatment is rarely urgently needed, but should, they say, be unhesitatingly employed in acute cases, in cases tending rapidly towards cachexia, and where the thyroid is giving rise to signs of compression. In the majority of cases the attack will be along several lines concurrently, isolation, hydrotherapy, psychotherapy, electricity, and serotherapy perhaps all being employed at the same time. J. H. HARVEY PIRIE.

A MIND THAT FOUND ITSELF: AN AUTOBIOGRAPHY.

CLIFFORD WHITTINGHAM BEERS. London: Longmans, Green & Co., 1908, Pp. 363. Price 7s. 6d. net.

WHATEVER the result of this book may be with regard to the object which the author has so much at heart, there can be no question that it is full of interest to the student of mental disorder and to every one engaged in the care of the insane. It is a very full and detailed record by the author of his own case and of his experiences during three years of treatment in various institutions in America. For over two years he was depressed and full of morbid suspicions. He then abruptly, after one link in his chain of morbid ideas was demolished, experienced a complete readjustment, but passed into a state of marked elation with continual activity and great over-productivity both in speech and writing. After his recovery from this second phase he had one later attack. The patient was treated in a Sanatorium, a Private Hospital, and a State Hospital for the Insane, and the details of the treatment and the effect of the treatment upon him at the time are the main theme of the work. The flaws in the care of the insane, the unsatisfactory nature of the attendants in some cases, the evil effects of the old-fashioned strong rooms and artificial forms of restraint are insisted on. But, while the patient on recovery felt bitterly with regard to certain episodes in his treatment, the book is written with moderation, and with a serious desire to do away with certain abuses which are only tolerated in the present day owing to the neglect of the public and owing to the deadening effect of routine on the administrative mind. The presentation of the subject may naturally be somewhat coloured by the personality of the author, and several additional facts might be added in order to adjust the perspective, but the book is one which cannot but be of use to any one who has to care for patients suffering from mental disorder. It is written especially for the American public and deals with the local situation, but it has an important lesson for a country like Great Britain, where, in a large number of the asylums, there is no

adequate provision for the treatment of cases of excitement, and where, instead of the continuous bath, the physician has the alternative of employing either chemical restraint or padded rooms, treatment now recognised on the Continent to be obsolete.

The author does not minimise the practical difficulties in the way of a solution of the question, and in the second half of the book he brings forward suggestions as to practical methods of remedying existing evils. There are five appendices: "Mechanical Restraint and Seclusion of Insane Persons," by Charles W. Page; a report of the American Consul-General at Berlin, entitled "Modern Hospital for the Insane," describing the new psychiatric clinic in Munich, the most modern in Germany; official statistics of "Insane and Feeble-Minded in Hospitals and Institutions, 1904"; a description of the working of the Pavilion for Mental Diseases in the Albany Hospital; certain strictures by Professor Allen Starr on nursing in private insane asylums.

C. MACFIE CAMPBELL.

A MEDICO-PSYCHOLOGICAL STUDY OF ALFRED DE MUSSET.

(*Étude médico-psychologique sur Alfred de Musset.*) R. ODINOT,
Thèses de Lyon, 1906-1907. No. 13.

THIS thesis, inspired by Professor Lacassagne, contains an interesting study of the celebrated poet. The morbid character of Musset's genius is illustrated both by the remarks of his contemporaries and by quotations from his works. His heredity was good, but from infancy he was of an extremely irritable and melancholy disposition. Odinet describes Musset as a superior degenerate, characterised by impulsiveness, instability, aboulia, and obsessions. In this degenerate soil symptoms of hysteria and neurasthenia developed. Musset commenced his first alcoholic excesses at puberty and continued them through life. He also indulged freely in opium and tobacco. During his stay at Venice with George Sand he contracted malaria, which was probably the cause of his subsequent deafness, auditory hallucinations, and aortic incompetence. Though frequently exposed to infection, he does not appear to have contracted syphilis. His moral and physical decadence dated from ten years before his death, being determined by excesses of all kinds, and not least by the "Green Muse" of absinthe, from whom he had sought consolation and inspiration.

His death at the early age of forty-seven from aortic incompetence was preceded for some years by anginal attacks, for which opium was freely taken. A remarkable symptom of his aortic disease during the last few years of his life was a jerk of the head corresponding to each cardiac systole. This phenomenon has since been known as Musset's sign.

J. D. ROLLESTON.

CROWD SUGGESTION AND PSYCHICAL CROWD EPIDEMICS.

(Über Massensuggestion und psychische Massenepidemien.)

Professor HANS GUDDEN, Verlag der Aertzlichen Rundschau, München, 1908. M. 0.75.

THIS is a popular lecture detailing some of the better-known facts and instances of crowd suggestion. The psychological differences in crowd behaviour and individual behaviour are pointed out—rather on the lines of Sidis' work on the psychology of suggestion—but the origin and significance of them is not inquired into. The volume contains nothing—beyond a few recent examples—that may not be found fully discussed in the classic work of Le Bon.

ERNEST JONES.

ZUR PSYCHOLOGIE DES FALLES MOLTKE. GEORG MERZBACH

(of Berlin). Berlin: A. Hölder, 1907-8. Pp. 44.

THIS brochure is one of the products of the late somewhat unsavoury case of Harden-Moltke which created so much interest in social and political circles in Germany. The author, a personal friend and medical adviser of Moltke's family, was not permitted in Court to make any complete statement on the matter at issue. He therefore takes this opportunity to lay before the public his conviction that Count Moltke was quite free from homo-sexual tendencies. He gives a slight sketch of the facts upon which he bases his views, and analyses the psychopathic personality of the divorced wife of the Count. The work is interesting from the medico-legal point of view, but has no wider bearing than the elucidation of the individual case.

C. MACFIE CAMPBELL.

BOOKS AND PAMPHLETS RECEIVED.

Wm. A. White. "Outlines of Psychiatry." New York, 1908, \$2.00.

Max Kauffmann. "Beiträge zur Pathologie des Stoffwechsels bei Psychosen. Erster Teil: Die progressive Paralyse." Fischer, Jena, 1908, M. 6.

"Neuropathological Papers," 1906 and 1907. Harvard University Medical School.

Fabritus. "Studien über die sensible Leitung im menschlichen Rückenmark auf Grund klinischer und pathologisch-anatomischer Tatsachen." Karger, Berlin, 1907.

Tycho Tullberg. "Linnéporträtt. Vid Uppsala Universitets Minnefest på Tvåhundraårsdagen af Carl von Linné." Stockholm.

"Department of Neurology; Harvard Medical School," vol. iii. Boston, Mass., 1908.

Review of Neurology and Psychiatry

Original Articles

A NOTE ON AN ASSOCIATED MOVEMENT OF THE EYES AND EARS IN MAN.

By S. A. K. WILSON, M.B., B.Sc., M.R.C.P.,
Registrar to the National Hospital, Queen Square, London.

SOME time ago, in the course of an examination of a case of syphilitic paraplegia in the National Hospital, under the care of Sir William Gowers—who has kindly allowed me to refer to it—my attention was drawn to a curious and constant phenomenon exhibited by the patient. As I was testing the lateral movements of his eyes, I noticed that when the latter were strongly deviated to one or other side the helix of the homolateral pinna curled backwards slowly and deliberately, chiefly in its middle part, just as the eyes attained the extreme position, and that it resumed its normal place as the eyes returned. This movement was constant in its appearance, and easily demonstrable, and it was shown to various members of the Staff of the Hospital, who corroborated my observation. I found that the heterolateral ear behaved similarly, though in its case the posterior edge of the pinna did not curl quite so far round. When the patient looked to the right both ears moved in this way, the right more than the left, and *vice versa* when he looked to the left. There was no movement of the pinna as a whole, *i.e.* the extrinsic muscles of the ear were not concerned; what

happened was that the helix curled towards the cranium and, when viewed from the front, partially disappeared behind the antihelix. The patient was unable, nor had he ever been able, to move his ears voluntarily, as some can. Further, he was unconscious of the movement that I have been describing.

If the accompanying photographs of the patient are examined carefully, it will be seen that in Fig. 1, which is a full face view, the outer edge of the right helix is opposite a point on the scale which corresponds to $8\frac{1}{8}$: it is between the $8\frac{1}{2}$ and the $8\frac{3}{4}$ marks. The helix of the left ear is opposite a point between the marks for $17\frac{1}{8}$ and $17\frac{1}{4}$, being rather nearer the former: it will suffice if we say it corresponds to $17\frac{3}{8}$. When the patient turns his eyes conjugately to the right, as in Fig. 2, his head remaining immobile, we see at once that the contour of each pinna has changed. The outer edge of the right helix is now opposite a point corresponding to $8\frac{1}{2}$, while the left helix is opposite 17. According to this measurement the upper part of the homolateral helix has curved back about $\frac{1}{8}$ of an inch, while the heterolateral helix has moved through about $\frac{3}{8}$ of an inch. As a matter of fact, however, the scale has been fastened a little too high, for a glance at the figure will show that the lower part of the right helix has moved through a space of considerably more than $\frac{1}{8}$ of an inch, and has disappeared behind the antihelix, while the left helix has moved rather more than $\frac{3}{8}$ of an inch, but still not so much as its fellow of the homolateral side. A photograph taken with the eyes deviated to the left shows a similar condition, the left ear having moved rather more than the right.

The movement of the ears in this patient's case having presumably nothing to do with the disease from which he suffered, I examined a series of fifty individuals (men) from the point of view that concerns us here. No one knew beforehand the object of the investigation, and all, as far as could be ascertained, were free from any cranial nerve defect. Many were in a state of normal health. The method was simply as follows:—The individual was seated with his head resting on some convenient support and kept strictly in the mid-plane, either voluntarily or steadied by my hand. He was then asked to look as far as he could to one or other side; sometimes he was directed to follow the tip of my finger as it was moved round.

Of these 50 individuals, 30, or 60 per cent., showed no



FIG. 2.

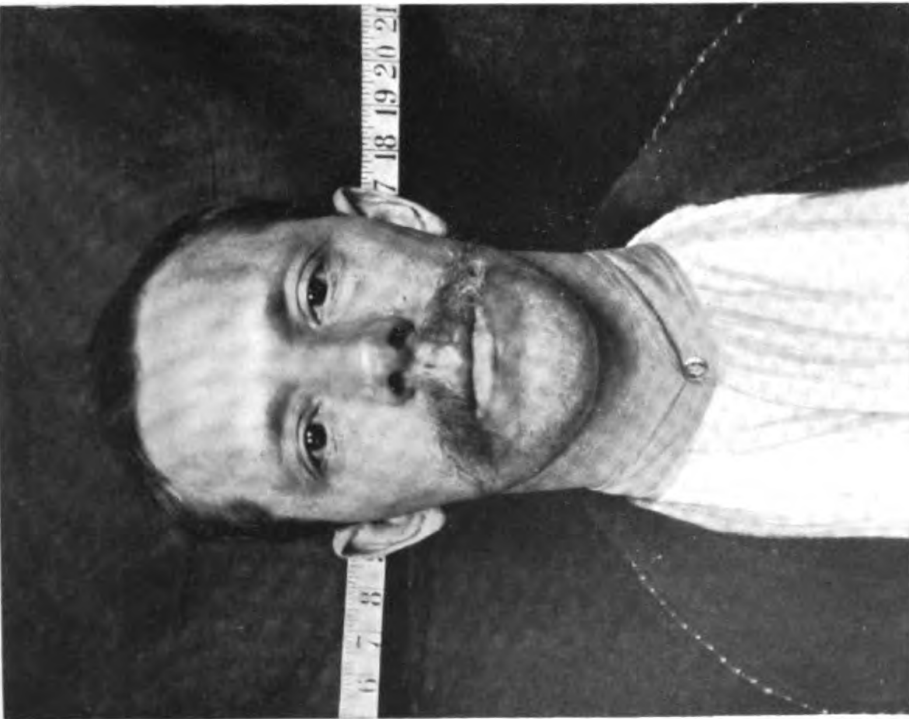


FIG. 1.

movement of the pinna that could be detected. The other 20 did exhibit some movement ; 6 gave a movement of the right ear on looking to the right, and of the left ear on looking to the left (in 2 of these cases the movement of the left ear was obviously greater than the movement of the right) ; 5 gave a similar homolateral movement from either side, and in addition showed a synchronous heterolateral movement which was *greater* than the homolateral movement (in one of these cases the curling back of the pinna of the left ear when the subject looked to the right was more evident than that of the right ear when he looked to the left ; in another exactly the opposite condition was noted) ; 2 showed a similar state to the 5 above mentioned, except that the synchronous heterolateral movement was *less* than the homolateral movement ; 5 gave a movement of one homolateral helix only (in 4 of these it was the left ear) ; one revealed only a heterolateral movement, the right ear curling when the eyes were directed to the left ; and one gave a homolateral and a heterolateral movement from one side only, viz., when he looked to the left.

Thus in no fewer than 40 per cent. a movement of one or other ear was noticed, and in 13 of the 20, or 26 per cent. of the series, a homolateral movement was obtained from either side.

In none of the subjects examined was there a movement of the pinna as a whole ; and though some were able to move their ears voluntarily, this power bore no relation to the occurrence of the particular movement under consideration ; further, none of them could reproduce voluntarily this peculiar phenomenon, which consisted, as has been said, in a curling movement of the helix and posterior part of the pinna about its middle towards the side of the head.

It is, I think, clear that this deviation of the helix must be due to contraction of the transversus auriculæ muscle, which is placed on the cranial surface of the pinna, and consists of scattered fibres, partly tendinous and partly muscular, extending from the convexity of the concha to the prominence corresponding with the groove of the helix (Gray). In its contraction it probably tends to open the orifice of the meatus ; and it is the homologue of an intrinsic ear muscle which possibly attains a greater development lower in the scale of animal creation.

That this little muscular sheet, and it alone, should be

innervated in association with lateral deviation of the eyes seems to me to be a curiously interesting fact, and yet it is not more so than other associated movements with which we are familiar. If we close the eyes tightly we often are aware of a low rumbling sound in the ears which, in all probability, is not the muscle sound of the contracting orbiculares, but is an associated movement of the tensor tympani. The orbiculares are supplied by the seventh cranial nerve, while the tensor tympani is innervated by a motor filament from the otic ganglion probably coming from the facial by the small superficial petrosal. When the eyelids are shut the eyeball rolls upward in association; the superior rectus and the inferior oblique receive their supply from the oculomotor nerve. Sometimes the opening of the mouth is associated with a synchronous elevation of the upper eyelid, and the assumption has been made that there must be twigs from the motor fifth going to the levator palpebræ superioris.

In a consideration of these and other instances that might be cited, we should direct attention to the association of *movements* rather than the association of *muscles*. There is no justification for the supposition that when certain of the muscles in a particular nerve distribution are innervated, some neighbouring minute nerve filament going to some insignificant muscle possesses some special property whereby it is inevitably innervated at the same time. We gain a truer conception of the significance of the elevation of the eyelid with opening of the mouth, or of the upward rotation of the eyeball with closure of the eye (4), if we consider the association as one of movement and not of muscles. Let us bear this in mind in the discussion of the associated movement which is the subject of this paper.

There is no obvious anatomical explanation of the association in contraction of a minute muscle, supplied by a correspondingly minute twig of the seventh, with a pair of other muscles acting in unison under an impulse from the sixth nerve nucleus or a supranuclear centre. And not only so; there is no necessity even to seek an anatomical reason when we remember the general principle that anatomical juxtaposition does not involve functional co-operation. We must look for a clue to the phenomenon in a physiological and ontological direction.

It has long been thought that in the anterior and posterior colliculi we have a centre, at least in animals, for certain

combined movements of the eyes and ears. On stimulation of the anterior corpora quadrigemina in monkeys, Ferrier (2) found that "irritation of the one side causes the opposite pupil to become widely dilated. . . . The eyeballs are directed upwards and to the opposite side. . . . The ears are strongly retracted. . . . Irritation of the posterior tubercles causes the same . . . general motor symptoms." Bechterew (1) records: "Es ist interessant hier zu vermerken dass Ich zuweilen nach der einseitigen Verletzung des hinteren Zweihügels bei Kaninchen beobachten konnte dass das Thier in der ersten Zeit nach der Operation die contralaterale Ohrmuskel nicht aufrichtete. . . ." And, on stimulation, "Die Augäpfel werden vor allem nach der entgegengesetzten Seite hin abgelenkt und die gegenüberliegende Ohrmuskel nach aussen gedreht und aufgerichtet." Prus (5) found that from various parts of the anterior colliculus he could make the ears turn in various directions, and the eyes move conjugately to the opposite side, and he noted, in addition, that these movements of the eyes and ears persisted on stimulation, even though the surface of the colliculus was cocainised. The assumption, therefore, is that they are the direct result of the stimulation, and not reflex in origin. From the posterior colliculus he obtained deviation of the eyes to the opposite side, but no ear movements. On the other hand, Thiele (6) states that "stimulation of the lateral aspect of the posterior corpora quadrigemina gave rise to no movements of the eyes. . . . Stimulation of the fillet, however, gave rise uniformly to pricking and turning back and down of the contralateral ear, and rotation of the eyes to the contralateral side — the movements . . . being of a similar nature to those that would naturally occur from the stimulation of the ends of the contralateral auditory nerve, the animal, of course, turning its head and eyes to the side from whence the sound came, and pricking the ear towards it to localise it." Sir Victor Horsley (3) also has referred to this point: "I have made many observations on excitation of the colliculi, anterior and posterior . . . and in the cat, dog, and monkey have observed . . . that faradic excitation of the outer surface of the posterior colliculus evokes . . . marked rotation of the pinna of the contralateral ear. . . . Not infrequently the homolateral rather than the opposite ear moves, but the character of the movement is the same, viz., rotation for

better audition. . . ." The close propinquity of the fillet to the corpora quadrigemina makes it possible that the results of earlier experiments are, in part at least, attributable to spreading of the stimulus to the former structure.

It is patent, both from experimentation and clinical observation, that in many animals a functional association of the eyes and ears is frequent; conjugate deviation of the eyes to one side is accompanied by pricking and rotation backward of the corresponding ear—an association of movement which is obtainable experimentally by stimulation of centres superior to the lower neurones. It is as though the animal makes this movement or movement-complex in response to an auditory stimulus; the eyes look in a certain direction; the ear is pricked or rotated to catch a sound. The action of the transversus auriculæ, as has already been said, is one which tends to open the orifice of the meatus, and thereby to aid audition; and it is therefore not improbable that the universal habit of turning the eyes in the direction of a faint sound is no meaningless movement, but a method, unconscious though we be of it, for increasing our chances of catching the sound by enlarging the mental opening. I venture to suggest that in this functional connection of the transversus auriculæ with conjugate deviation of the eyes we have a remnant of an associated movement that is of widespread occurrence among the lower vertebrates. It is conceivable that individuals in whom the phenomenon is not forthcoming may not have the intrinsic ear muscles adequately developed. In the anthropoid apes the transversus auriculæ is well marked, but below them little definite is known as to the development it attains.

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FIG.



FIG. 1.

**A CASE OF PARTIAL PTOSIS WITH EXAGGERATED
INVOLUNTARY MOVEMENT OF THE AFFECTED
EYELID : The "Jaw-winking" Phenomenon.**

By WILLIAM GEORGE SYM, M.D.

THE features of this case are these: A. J., a young woman of 30, came to me from the North of England complaining of two things, namely partial ptosis on the left side, and secondly (and from her point of view more importantly) that at times the left upper lid executed involuntary and "ridiculous" movements. On examination I found that the only fault to be found with either eye was that which is well illustrated in the two accompanying figures (Pl 19, figs. 1, 2). There was a very distinct degree of ptosis of the left upper eyelid, the levator of which, however, was by no means paralysed, for within limits it enjoyed ready and free movements. Movements of the globe were perfect; there was not a trace of halting or failure to look upwards or in any desired direction. When, however, the patient was chewing her food or when she was singing, the left upper lid, one might almost say, sprang upwards, disclosing perhaps as much as 3 mm. of white sclerotic above the corneo-scleral junction. It was this really which had distressed the patient so much, for she could not take food in the presence of strangers without remarks being made upon the curious appearance of her eye, nor could she, though very fond of music, sing before others, for the same reason. I exhibited the patient to Dr Bruce as an example of what has been—very inelegantly—called the jaw-winking phenomenon, and together we investigated the case further.

There was very distinct elevation of the eyelid on use of the left external pterygoid muscle (moving the jaw to the right), but it was merely a partial lift, a sort of exaggerated twittering of the eyelid up and down; when, however, she suddenly opened the mouth by actively depressing the lower jaw, the lid sprang up in the manner shown in the second photograph. There was at the same time no exophthalmos or any alteration of the pupil; vision was good and equal. When the lid flew up there was no corresponding movement of the right upper lid, there was no corrugation of the forehead, nor was there any untoward movement of the eye itself, whether lateral, vertical, rotatory, or antero-posterior. Nor was

there any evidence which might point to any affection of the sympathetic on one side, such as unilateral sweating or flushing. The condition had been present all the patient's life; she could not say there was any change from childhood. She had no sensation of anything happening; it was the obvious, unpleasing facial contortion which had brought her, nothing else.

The first case of the kind was described by Marcus Gunn in 1883, and since then some twenty to thirty cases have been noticed. Mr Gunn showed his patient at a meeting of the Ophthalmological Society, a committee of the members of which afterwards reported upon the case. The patient was a girl who presented very similar symptoms to those of A. J., only that the involuntary movement of the eyelid came on rather with lateral movement than with vertical opening of the mouth. The committee which investigated the case came to the conclusion that probably the levator was innervated partly from the nucleus of the third nerve and partly from the external pterygoid portion of the nucleus of the fifth nerve, and that there was an abnormal connection between the two. If some of the innervation of the lid (which is evidently defective) arose from the fifth nucleus, that would account for the appearances.

Much the most important paper written on this subject will be found to be that of Sinclair of Ipswich, who in 1895 collected a number of cases of associated movement, and reviewed them in the *Ophthalmic Review* for October of that year. These he divides into three series, of which the first consists of those in which certain movements of the lower jaw are associated with an upward movement of the eyelid; the other two series do not concern us at present; they are of quite a different type. Our present case would fall to be included in his first group under Series I, for that includes cases in which either lateral movement or wide opening of the mouth brings on the elevation of the eyebrow. The second group takes in the cases in which depression of the lower jaw alone brings on the elevation; in the third group are those cases in which lateral movements of the jaw alone, as in the process of chewing, set up the contraction, but vertical movements do not. In some few of the cases the untoward movement only took place when the eye was also looking down; that was not true of the present case. It is a curious fact that out of twenty-five cases in which the point is noted, the error was on the

right side only in seven; in all the others it was left-sided. Although ptosis was present in nearly all, the associated movement was noticed in a very few without any ptosis existing. It has often been remarked upon that if one endeavours to induce a child to open the eyes widely he is very apt to open his mouth instead or in addition, and all the more so should there be any blepharospasm at the time. Ole Bull and others consider that such a movement is simply reflex, and that the special form we are discussing is analogous to it. But Sinclair is perfectly correct when he points out several objections to such an idea, particularly that in some cases it is on lateral movements of the jaw only that the symptom occurs, that the symptom is one-sided only, and that in the children spoken of the attempt to elevate the lid is carried on by the frontalis muscle, and the facial aspect is therefore entirely different. The explanation offered by the committee on Gunn's case is much more satisfactory, namely, that the levator receives nerve impulses both from III. and V. nerves. Helfreich has suggested that the abnormal fibres might perhaps come from the facial. In a certain number of the cases it may be true, as suggested by some, that there may be imperfect development of the oculo-motor nucleus; but, as we have just mentioned, ptosis is not invariably present.

A singular paper on the subject has been written by Harman, who endeavours to explain the association of lid-movement along with jaw-movement, "not as a 'freak,' but as a revival of an old-time and long-accustomed associated movement." He traces the facial musculature back to our ancestors the fishes, where it forms the spiracular musculature, the pterygoid muscle of man being the lineal descendant of the deep gill-muscle which moves the maxillary cartilage. He considers that as the eye, in the process of development, became a more mobile organ than primitively it was, it filched from the spiracle its muscular apparatus, and thus in man a case will now and then turn up in which this atavistic association between jaw on the one hand and spiracle-modified-into-mobile-eye on the other is manifested. His conclusions are not, it must be confessed, very convincing.

To sum up, there does not appear to be any other probable explanation of this curious association than that in some inexplicable way there arises some confusion in the joining up of fibres and cells belonging to the fifth and third nuclei in such fashion

that the levator receives less than its normal innervation, and there is therefore a certain degree of ptosis (though this is not a necessary part of the error), but there is no paralysis of the muscle, which is capable of full contraction ; at the same time, the levator receives some fibres which were "intended for" the external pterygoid or the digastric, and when that muscle is put in action, at all events when put strongly in action, the levator is unintentionally innervated, producing the curious effect described. When examining the present case along with me, Dr Bruce put forward tentatively the suggestion that the elevation of the lid might be caused by undue contraction of the unstriated muscle of the lid, and it must be admitted that the appearance of the eye is not unlike the familiar aspect in exophthalmic goitre ; but I think myself that the contraction is too sudden and swift to be due to the unstriated muscle, and the unusual nature of the aspect is due to the fact that we never see the levator act under normal conditions without the elevator muscles of the globe. Thus we never see that gap of sclerotic between the upper limit of the cornea and the edge of the upper lid which is so well brought out in the photograph as occurring in this patient. We should take along with this case, too, those others in which *other* muscles supplied by the third nerve have had their innervation mixed up with that of the jaw muscles, causing the globe to take up an abnormal position or the pupil to contract.

Since 1895, when Sinclair analysed the cases published up to then, I am only aware of two other examples, those of Fischer and Harman, shown at the Ophthalmological Society in 1889 and 1903. The matter receives recognition in very little of the recently published literature dealing with the eye and the nervous system.

Abstracts

ANATOMY.

**THE DEVELOPMENT OF THE MAMMALIAN PITUITARY AND
(285) ITS MORPHOLOGICAL SIGNIFICANCE.** P. T. HERRING,
Quar. Journ. of Exper. Physiol., Vol. i., No. 1, p. 161,
April 1908.

IN mammals the epithelial portion of the pituitary is divided entirely from the ectodermic wall of the buccal invagination

known as Rathke's pouch. The epithelium is differentiated at an early stage into two parts. One, the pars intermedia, is closely adherent to the wall of the cerebral vesicle from its earliest appearance and remains attached to it throughout life. It tends to arrange itself over the adjacent surface of the brain in positions where it can approach as near as possible to the cerebro-spinal canal. The other portion of buccal epithelium situated in the lower portion of Rathke's pouch gives rise to the anterior lobe proper, and forms a solid mass of cells which grows into neighbouring blood-vessels and into the cavity of the pouch itself. Part of the cavity persists to a greater or less extent as the epithelial cleft.

The infundibulum is an invagination of part of the wall of the thalamencephalon which is adherent to the anterior and upper wall of Rathke's pouch, and possesses, therefore, an epithelial covering derived from the latter. The infundibulum with its covering becomes the posterior lobe of the pituitary.

The intimate nature of the connection between the wall of Rathke's pouch and the cerebral vesicle, and the maintenance of this close relationship throughout life, render probable the view adopted by Kupffer that the pituitary body of mammalia is to be regarded as the representative of an old mouth opening into the canal of the central nervous system. Such an arrangement exists in its simplest form in the Ascidian larva. A connection is sometimes seen in the developing cat between Rathke's pouch and the interior of the infundibulum, these cavities being continuous for a short period of foetal life.

AUTHOR'S ABSTRACT.

"THE HISTOLOGICAL APPEARANCES OF THE MAMMALIAN (286) PITUITARY BODY." P. T. HERRING, *Quar. Journ. of Exper. Physiol.*, Vol. i., No. 2, April 1908.

THE pituitary differs widely in shape and in the arrangements of its constituent parts in various mammals. In all there is a solid glandular lobe permeated by wide blood channels or sinusoids, and an intermediate part of epithelial cells closely connected with a third portion which consists of nervous tissue derived from the brain. The pituitary bodies of mammals may be roughly divided into three types, according to the degree in which the infundibular cavity is prolonged into the nervous lobe. In the cat the cavity runs almost to the posterior margin of the lobe, in the dog it stops short in the neck, and in man, monkey, and other animals examined the neck and posterior lobe are usually solid, although a narrow lumen is sometimes continued into them.

The anterior lobe consists of columns of cells, some of

which are clear, others granular and deeply staining; the latter give to the lobe its characteristic appearance. These cells are probably functional stages of the same kind of cell, and the granules represent the material which is passed into the blood-vessels as an internal secretion. No colloid occurs in the anterior lobe proper.

The intermediate portion is made up of finely granular epithelial cells, closely applied to the body and neck of the nervous lobe and to the adjacent part of the brain. That part of it which covers the body of the posterior lobe is separated from the anterior lobe proper by a cleft, and is almost devoid of blood-vessels. In the cat the portion lying in front of the anterior lobe has a tubular appearance and is very vascular. Colloid material is commonly met with throughout the pars intermedia, and, in most situations, appears to pass into the adjacent nervous substance.

The nervous portion consists of neuroglia cells and fibres, and contains no true nerve cells. Large ependyma cells line the central cavity in the cat and send long fibres forwards and upwards towards the brain, most of which terminate in the outer part of the neck. These ependyma fibres are numerous and coarse, and give to the neck of the lobe an appearance as though of nerve fibres running longitudinally in it. Epithelial cells of the pars intermedia surround and invade the nervous substance; islets of them are not uncommonly seen, and may even lie within the central cavity. A substance resembling colloid is found in relation to the epithelial cells, and occurs widely throughout the lobe. It is especially large in amount towards the neck of the lobe, and may be seen among the ependyma cells passing into the central cavity and so into the third ventricle of the brain. This part of the pituitary body may be regarded as a brain gland, the cells of the pars intermedia furnishing the secretion.

The pituitary body probably receives its nerve supply through the sympathetic system. The anterior lobe is extremely vascular; the posterior lobe has a smaller and separate blood supply, with a capillary circulation unlike the sinusoids of the anterior lobe.

From a physiological point of view the pituitary body consists of two distinct parts, in both of which the epithelium is the active agent. The anterior lobe is an internally secreting gland, the function of which is unknown. The posterior lobe has a framework of neuroglia and ependyma supporting epithelial cells, which apparently produce a secretion destined to enter the ventricles of the brain. Extracts of this lobe are in some respects similar in action to extracts of the medulla of the suprarenal capsule, but have

in addition a selective action upon the kidney causing dilatation of the renal blood-vessels and diuresis. Disturbances of the posterior lobe are probably responsible for the occurrence of the diabetic conditions which have been so frequently recorded at some stage or other in the history of cases of acromegaly and of affections and lesions associated with the base of the skull.

AUTHOR'S ABSTRACT.

ON THE NEUROFIBRILS IN THE PROCESSES AND THE CELL-BODY OF MOTOR GANGLION CELLS. (Über das verschiedene Verhalten der Neurofibrillen in den Fortsätzen und dem Zelleib der motorischen Ganglienzellen.) GIERLICH, *Neurol. Centralbl.*, Dec. 16, 1907, p. 1154.

FROM the investigation of various sections prepared by Bielschowsky's method, the writer found that during the development of motor cells the neurofibrillæ appear first in the dendritic processes and then later in the cell-body; and that in degeneration the fibrils of the cell-body undergo disintegration earlier than those of the processes.

ALEXANDER BRUCE.

THE POSTERIOR LOBE OF THE PITUITARY GLAND. (Le lobe postérieur de la glande pituitaire.) H. JORIS, *L'Acad. roy. de méd. de Belgique*, Tome xix., f. 10.

JORIS now finds from the results of his comparative anatomical and embryological investigations that the posterior lobe of the hypophysis is not a degenerated structure, but is an active gland secreting a substance which has been found to modify the blood pressure and diminish the number of the heart beats whilst augmenting them in force.

If this structure represented, as some believe, in the higher vertebrates the ruins of a nervous centre, it should be found completely developed in lower vertebrates, whilst if it represented an atrophied gland it should appear as a developed gland in lower vertebrates. To test this point he has examined a large number of hypophysi from fishes among whom it attains an extreme degree of development, and also from higher vertebrates (cat, dog, guinea pig) at various stages of embryological development; and finds that in all these cases it appears as a glandular formation of mixed origin, derived partly from the infundibular and partly from the pharyngeal prolongation.

JOHN TURNER.

PHYSIOLOGY.

THE PHYSIOLOGICAL ACTION OF EXTRACTS OF THE
 (289) **PITUITARY BODY AND SACCUS VASCULOSUS OF**
CERTAIN FISHES. P. T. HERRING, *Quar. Journ. of*
Exper. Physiol., Vol. i., No. 1, April 1908, p. 187.

THE pituitary body of elasmobranchs, *e.g.* the skate, has no posterior lobe, neither does its anterior lobe possess the granular cells which are characteristic of the anterior lobe of higher vertebrates. Extracts injected intravenously produce in the cat a slight fall of blood pressure, a dilatation of the kidney and some increase in the flow of urine; the latter is so slight that it is doubtful if it is due to a specific action.

In teleosts, *e.g.* the cod, the pituitary body more closely resembles that of mammals. Its anterior lobe contains the deeply staining granular cells, and there is a nervous portion surrounded and invaded by clear cells resembling those of the pars intermedia of mammals. Extracts of pars intermedia and pars nervosa produce the typical effects of extracts of the posterior lobe of the mammalian pituitary.

Extracts of the saccus vasculosus of the skate or cod have no marked physiological effect when injected; very strong extracts give practically no effect other than that of the Ringer's fluid in which they are made up. The saccus vasculosus was considered by Gentes for anatomical reasons to be a ventral choroid plexus, and the inactivity of its extracts tends to support this view.

AUTHOR'S ABSTRACT.

STUDIES IN RESUSCITATION — II. THE REFLEX EXCIT-
 (290) **ABILITY OF THE BRAIN AND SPINAL CORD AFTER**
CEREBRAL ANÆMIA. F. H. PIKE, C. C. GUTHRIE, and
 G. N. STEWART, *Amer. Journ. Physiol.*, Vol. xxi., No. iii.,
 p. 359.

THE head arteries were temporarily occluded in cats by ligature or clamp, depriving the brain and the upper part of the spinal cord of blood supply, and the reflexes were examined during occlusion and after release of the vessels.

The anterior part of the cord becomes totally inexcitable during the period of anæmia, but may regain its excitability more or less completely when the circulation through it is re-established, and may for a time become hyper-excitable. This hyper-excitability affects also the posterior part of the cord. In some cases the anterior part of the cord never regains completely its normal excitability.

Many of the phenomena of spinal shock may be produced by this method, and when the period of anæmia has been sufficiently prolonged to cause severe injury to the encephalon and anterior part of the cord, the animal falls into much the same condition as the "spinal" or "bulbo-spinal" animal, and the scratch reflex is easily elicited.

SUTHERLAND SIMPSON.

PATHOLOGY.

ON NEUROTOXIC SERA AND THE LESIONS OF THE NER-
(291) VOUS SYSTEM CAUSED BY THEM—ISONEUROTOXIC
SERUM. (Contributo allo studio dei "Sieri neurotossici"
 e delle lesioni da essi provocate nel sistema nervoso—
 Siere isoneurotossico.) O. ROSSI (of Florence), *Riv. di Pat.*
nerv. e ment., Sept. 1907.

THE injection into an animal of given species of a preparation of the nervous tissue of an animal of another species confers on the serum of the former toxicity with regard to the nervous system of the second. This observation can be made by injecting dog brain into guinea-pigs and then injecting the serum of the latter into the dog. The clinical manifestations vary according as the grey or white matter is used. After several injections of guinea-pig brain into the guinea-pig, the serum of the latter acquires a toxicity for the nervous system of the guinea-pig. The action of these sera is not at all specific; in the author's experiments the isoneurotoxic serum produced pathological phenomena in the dog as well as in the guinea-pig. The properties of these sera do not agree in all points with those of the typical cytolytic serum, *i.e.* the hæmolytic serum. The anatomical changes are constant and well marked; the most severe are those implicating the nervous tissue.

C. MACFIE CAMPBELL.

ON CHANGES IN THE PERIPHERAL NERVES IN GENERAL
(292) PARALYSIS AND OTHER PSYCHOSES. (Beiträge zur
 Kenntnis des Vorkommens von Veränderungen in den
 peripheren Nerven bei der progressiven Paralyse und ein-
 zelnen anderen Psychosen.) E. STRANSKY (of Vienna), *Arb.*
a. d. Neur. Inst. a. d. Wien. Univ., 1907, p. 281.

THE peripheral nerves in 60 cases were examined by the Marchi method—general paralysis (29), cerebral syphilis (1), senile dementia (8), arteriosclerotic dementia (4), senile melancholia (1), senile mania (1), paranoia (4), dementia præcox (3), epilepsy (2),

amentia (2), alcoholism (5). In general paralysis parenchymatous changes in the peripheral nerves are on the average more frequent and more severe than, *ceteris paribus*, in other mental disorders accompanied by marasmus and somatic complications.

C. MACFIE CAMPBELL.

LESIONS OF THE CEREBRAL AND CEREBELLAR CORTEX IN (293) ALCOHOLICS, WHO HAVE DIED SUDDENLY FROM ACCIDENT OR HOMICIDE. (Lesioni della Corteccia Cerebrale e Cerebellare in Individui Alcoolisti, etc.) V. ALESSI, *Ann. di Neurologia*, Anno xxv., f. 4-5, 1908.

THE author of this paper holds a position in which he has exceptional opportunities of investigating the nervous systems of individuals who are suddenly killed while in a state of alcoholic intoxication. He records here the results of his investigation of five such cases.

The original paper is worthy of perusal, because a detailed account of the microscopical appearances is there given. In the cerebral cortex, the signs of degeneration are most marked in, and almost limited to, the layer of pyramidal cells; little change can be detected in the more superficial zones. Still more marked are the signs of degeneration in the cells of Purkinje in the cerebellar cortex; and the author correlates this fact with the clinical experience that inco-ordination is a prominent symptom of alcoholic intoxication.

The signs of degeneration referred to are chromatolysis of somewhat characteristic distribution, dislocation and vacuolation of the nucleus, varicosity of the protoplasmic processes, etc. The blood-vessels of both the cerebral and cerebellar cortex are distended with blood. There is no obvious affection of the neuroglia.

W. H. B. STODDART.

THE SENILE CEREBELLUM. (Sur le cervelet sénile.) ANGLADE (294) and CALMETTES (of Bordeaux), *Nouv. Icon. de la Salp.*, Sept.-Oct. 1907.

ON the basis of the examination of senile brains by the elective neuroglia method of Anglade, the authors conclude that the senile cerebellum presents characteristic lesions, and that the frequency and extent of these cerebellar lesions explain to a certain extent several of the symptoms in the clinical senile picture.

The senile cerebellum does not present an atrophy *en masse*, but rather limited patches of perivascular sclerosis; these patches

may occur anywhere in the cerebellum, but have certain points of predilection; there is considerable tendency to the formation of lacunæ. The patches of sclerosis are sharply defined, they are frequent in the neighbourhood of the cells of Purkinje, there is no meningitis associated with the process, and rod-cells are absent. The picture therefore is distinct from that of generalised cerebellar atrophy: there the sclerosis is diffuse, the cells of Purkinje absent, there are many nuclei and few fibres, the granular layer is indistinguishable from the white matter.

There is no confusion, therefore, between the purely atrophic and the senile cerebellum: one finds sometimes a patch of atrophic or hypertrophic sclerosis in the midst of senile lesions. The diagnosis of the senile cerebellum from that of the general paralytic is simple.

C. MACFIE CAMPBELL.

PSYCHOLOGY.

THE EFFECT OF MENTAL WORK ON AUDITIVE, VISUAL, (295) AND TACTILE SENSIBILITY. (Sur le mode de se comporter de la sensibilité auditive, visuelle, et tactile, à la suite du travail mental.) A. GRAZIANI (Padua), *Arch. Ital. de Biol.*, T. xlviii., F. ii., 1908.

DR GRAZIANI took advantage of a special course of lectures given by himself and two of his colleagues to men who were their equals in age and academic standing to study the effects of this considerable mental strain on the sensations of sight, hearing, and touch. The lecturers were tested in these respects—(1) on arrival at the laboratory, (2) immediately after the lecture, (3) after a rest of one hour. Qualitatively the results agreed in all three subjects, the changes observed being in the same direction; quantitatively, of course, individual differences appeared. The sensibility of the ear, tested by the ticking of a watch, was notably augmented after the lecture, the distance at which the sound was audible being increased by about 50 cm. After the hour's rest the sensibility had decreased to a little below normal. Analogous results were obtained with respect to the visual sensibility and the tactile sensibility of the face. With respect to the tactile sensibility of the hand no consistent variation could be established.

In Dr Graziani's opinion his results can be explained by two hypotheses based on already ascertained facts.

1. When mental work is being done an increased supply of blood comes to the brain; hence even the parts that are not working are in a state of hyperæmia. It may well be that this condition brings about an increase in the functional potentiality of

these parts. This supposition would explain the increased sensibility of eye, ear, and face. The fact that no increase of sensibility could be detected in the hand is obviously in favour of this explanation.

2. The investigations of Mosso with the ergograph, and the researches of numerous other authors, have shown that the first effect of mental or muscular work is to produce a period of excitation during which the output of work increases. Dr Graziani supposes that in virtue of the intimate connection which exists throughout the central nervous system, the whole may feel the excitation due to the work of a part, and thus a general increase in sensibility result. This hypothesis appears all the more probable in view of the fact that, according to the experiments of Griessbach the variations of tactile sensibility before and after mental work are really due to differences in the concentration of attention on the tactile sensation. The excitation produced by mental work not carried to the point of fatigue might very well increase this power of concentration.

These two factors, the one purely physiological, the other psycho-physical, appear to Dr Graziani necessary and sufficient to account for the phenomena observed by him. At the same time he emphasises the need for further observation and experiment.

MARGARET DRUMMOND.

CLINICAL NEUROLOGY.

PROGRESSIVE OSSIFYING MYOSITIS IN A BOY ÆT. ELEVEN.

(296) C. A. A. DIGHTON, *Edin. Med. Journ.*, April 1908.

THE condition started when the boy was five years old. The muscles became stiff, and following the stiffness hard lumps formed in the muscles, and have increased steadily up to the present time.

In the arms large nodules of the consistence of bone are felt in the biceps, triceps, coraco-brachialis, and anconeus muscles, the biceps being almost completely converted into bone.

In the lower limbs the adductors are completely converted into bone, while the extensors and flexors are affected to a less degree.

The case is typical in that the patient is of the male sex, that it is symmetrical, and that the disease commenced in early life. It is also quite the exception for the disease to have been in progress so long without affecting the muscles of the back.

An excellent skiagram is shown.

D. K. HENDERSON.

A CASE OF OLD MYOPATHY. (*Myopathie ancienne, etc.*)
(297) BALLET and LAIGNEL-LAVASTINE, *L'Encéphale*, March 1908,
p. 229.

THIS was a case of progressive myopathy, the patient dying at the age of thirty-seven. Clinically he presented the picture of a myopathy of the pseudo-hypertrophic type, affecting the shoulder and pelvic girdles, and progressing steadily until the muscular wasting was, in certain regions, extreme. There was a certain amount of generalised scleroderma, with the "facies de sphinx." Occasional fibrillary contractions occurred in certain muscles of the arms and neck, and at one stage a reaction of degeneration was obtained in them. Deep reflexes were abolished. The patient was mentally somewhat of an "arriéré." Pathologically changes of considerable interest were found. In the muscles, an exhaustive description of which is supplied, the lesions were those of a typical myopathy; involvement of individual fibres, some of which were markedly hypertrophied, others as markedly atrophied; the greater the hypertrophy the more translucent and vitreous the fibre, and the less evident its transverse striation. Surrounding the degenerating fibres was an interstitial sclerosis, with a certain amount of fatty change. Sometimes muscle fibres seemed to be disappearing in a sclerotic tissue rich in nuclei and strings of deeply staining round cells. Many of the fibres which were in an atrophic state showed an intensely basophilic formation in their interior of irregular shape, with prolongations losing themselves in the myoplasm. The appearance suggested a sarcoplasmic degeneration. The peripheral nerves furnished no indication of a neuritic process. Some were rather atrophic, specially such as were in connection with atrophic muscles.

In the spinal cord, in its cervical enlargement, the most noticeable feature was the almost complete absence of the anterior cornual cells: in a single section not more than one or two were discoverable, and these were confined to the antero-internal groups. Such cells as persisted were not deformed, but simply atrophic; there was no sign of chromatolysis, pigmentation, neurophagia or neuroglial proliferation. The vessels were normal. Sometimes traces of the cells were found in the shape of indefinite protoplasmic masses with a few chromatic grains within them. In ten sections the *total* number of anterior horn cells was seven for the antero-internal group, seven for the antero-external, and twenty-two for the lateral. In the motor cortex the Betz cells were usually globular and deformed, with excentric nucleus, but little pigment. By Pal's method the tangential fibres were seen to be rarified, while the fibres of Exner had almost disappeared in places. The large and small pyramidal cells seemed fairly normal, except that towards

the deeper parts of the cortex they were surrounded by considerable numbers of satellite cells, with an obvious increase of neuroglial elements.

A full discussion is appended of analogous cases in the literature. The authors' view is that the disappearance of the anterior horn cells is secondary to the atrophy of the muscular masses whose function they regulate. Attention is drawn to the concurrent mental changes.

S. A. K. WILSON.

SENSORY AND TROPHIC NEURITIS SECONDARY TO ZONA,
(298) **ETC.** (*Névrite sensitive et trophique à la suite d'un zona,*
etc.) ROSE, *Nouv. Icon. de la Salpêtrière.*, Jan.-Fév. 1908, p. 64.

IN a woman of sixty-seven an ordinary herpetic eruption occupying a strip down the outer side of the right arm was followed by violent pains in the shoulder, forearm and hand, and at the same time œdema of the forearm and hand and trophic changes in the bones made their appearance. The appearance of the hand resembled what one is accustomed to see in chronic rheumatism. The author supposes the osseous change to be secondary to the neuritis, which was in its turn a consequence of the original infection or intoxication.

S. A. K. WILSON.

A CASE OF PARALYSIS OF THE ABDUCENT AFTER SPINAL
(299) **ANÆSTHESIA.** (*Ein Fall von Lähmung des Abducens infolge*
von Rhachistovainisierung.) C. PARHON and M. GOLDSTEIN
(of Bucharest), *Spitalul*, No. 11, 12, 1907.

SPINAL anæsthesia has several advantages, but also many drawbacks, one of which is paralysis of the abducent some time after the operation. The real cause of this paralysis is not yet accurately known, nor has it been conclusively shown why the injection of an anæsthetizing solution into the arachnoidal space should act in an elective manner on the sixth pair. It is possible that it is a question of meningeal irritation or a toxic phenomenon.

The case observed by the authors was a man of thirty-one, who was operated on for hæmorrhoids under spinal anæsthesia; .1 cg. stovaine was employed. Ten days later there ensued complete paralysis of the left and paresis of the right abducent, which lasted more than three months.

E. TOFF (C.g.B.).

PARALYSIS CONSECUTIVE TO RACHISTOVAINISATION. (Con-
(300) tribution à l'étude clinique des paralysies consécutives à la
rachistovainisation.) MINGAZZINI, *Rev. Neur.*, March 15, 1908,
p. 185.

A YOUTH of sixteen was operated on for varicocele. A 5 per cent. solution of stovaine in a 5 per cent. solution of sodium chloride, acidified with lactic acid, was injected into the spinal theca. The amount injected is not specified. On the day after the operation the patient complained of headache, which continued for some hours and recurred on succeeding days. Thirteen days after the operation he observed that his left upper eyelid was drooping, and in the morning the eye was completely closed. On lifting the lid he saw double. On coming again under observation, a day later, it was found that his right eyelid also was drooping and that the right eye was deviated slightly outwards. In the course of nine or ten days the patient's condition scarcely altered: he was unable to look up with either eye; there was a certain amount of weakness of the left lower face, and fatigue soon showed itself in the lateral ocular movements. Two months later the diplopia persisted: there was a marked external strabismus of the left eye: the right ptosis was much more marked: upward and convergent movements of the eyes were weak. Pupillary reactions were normal, but better on the left side than the right. Gradually the patient became worse: the palate, orbiculares palpebrarum, and masseters became weak, and the limbs also felt rather feeble. There was no indication of muscular atrophy except to a slight extent in the left masseter. The voice was nasal. In short, the clinical picture bore a strong resemblance to that of myasthenia gravis.

Whether the stovaine played a part in the genesis of the condition is discussed at some length. Ocular palsies after stovainisation are not infrequent. The author inclines to the view that the toxic agent sets up a neuritic process in nerve trunks.

S. A. K. WILSON.

TABES DORSALIS AND ITS RE-EDUCATIVE TREATMENT.
(301) COLIN K. RUSSEL, M.D., *Montreal Med. Journ.*, Feb. 1908,
p. 90.

THIS paper is a *résumé* of Edinger's exhaustion theory of tabes dorsalis, showing its importance in the prophylactic treatment of tabes and its bearing on the re-educative treatment of Frenkel. Reference is made to Holmes' contribution to this theory.

AUTHOR'S ABSTRACT.

FAMILY SPASTIC PARAPLEGIA. (*La paraplégie spasmodique* (302) *familiale. Étude clinique.*) A. MENDICINI BONO, *Rev. de Méd.*, March 10, 1908, p. 209.

In this article the author gives short clinical notes of two cases of spastic paraplegia, and brief *résumés* of fifty-six other recorded cases. In his own cases there was in neither instance any other member of the family affected. In one, a man of thirty-nine, some stiffness in the legs had been noted since the age of twelve; there were no sensory symptoms, vesical symptoms had once been present but had disappeared, there was slight Rombergism; the spasticity, of slow development, was limited to the lower limbs. His second case, a woman of twenty-eight, had spasticity in the upper limbs also, and complained of shooting pains in the back and legs.

The author regards the disease as due to a degeneration of the pyramidal tracts in individuals who are poorly developed from some hereditary cause. There is frequently some associated slight lesion of the posterior columns. There is a good bibliography.

J. H. HARVEY PIRIE.

HÆMANGIOMA IN THE PONS VAROLII. NAMBU, *Neurol. (303) Centralbl.*, Dec. 16, 1907, p. 1162.

As hæmangioma is a rare form of brain tumour, the writer gives a description of one of the cavernous variety found in the case of a man of sixty-three. Microscopical examination showed that the tumour had no marked arterial or venous character, but was distinguished by the appearance in places of old thromboses of the blood spaces and by hyaline changes in the walls of the dilated vascular spaces. These spaces were lined with a distinct epithelium. Many of them contained blood, and others homogeneous masses, which were apparently old degenerated thrombi.

There was no clinical history of the case, but it is not likely that the tumour gave rise to any symptoms, as there was no secondary degeneration in the spinal cord.

ALEXANDER BRUCE.

THE SERUM TREATMENT OF EPIDEMIC CEREBRO-SPINAL (304) MENINGITIS. DUNN, *Boston Med. and Surg. Journ.*, March 19, 1908.

THE object of this paper is to enlist the co-operation of those in actual practice in testing the value of serum therapy in cerebro-spinal meningitis. In 1906 a number of favourable reports were published of the use of diphtheria antitoxin in this disease, but

Rotch and others have shown that no specific benefit is to be derived from such treatment. In 1907 Rotch treated cases with vaccines prepared from the meningococcus, but the results were totally inconclusive. Several investigators had meanwhile been working upon the production of a specific antiserum, and Flexner has brought such an antiserum into the domain of practical therapeutics. This antiserum is prepared from horses inoculated with the meningococcus, but it differs from diphtheria antitoxin in that its action is directed against the organisms themselves, and not against the toxins elaborated by the organisms, for which reason it is designated an antiserum and not an antitoxin. Flexner recommends that a maximum dose of 30 c.cm. should be injected directly into the spinal canal by lumbar puncture, and that the dose should be repeated daily for three or four days. The writer gives very full reports of fifteen cases treated by this antiserum. Of these, eight have recovered completely, having been left with no sequelæ of any kind; two have died, both chronic cases not seen till the disease had run for a considerable time; and five are still pending, four of them convalescent and certain to recover; one (a chronic case) doubtful. Early cases afford the greatest hope of permanent benefit from the treatment, and also exhibit the most marked response to the injections.

HENRY J. DUNBAR.

SOME CLINICAL OBSERVATIONS ON EPIDEMIC CEREBRO-
(305) SPINAL MENINGITIS. J. G. BROWNE, M.D., *Montreal Med.*
Journ., Feb. 1908, p. 98.

THIS report is based on 46 cases verified by bacteriological examination from the wards of the Montreal General and Royal Victoria Hospitals. The disease has been found to be more common in the spring and winter months, the largest number of cases occurring in May. Young adults in the second and third decades are most frequently affected, males 3·5 times more commonly than females.

The usual well-known symptoms have been noted at the onset and during the course of the disease. The following list of complications in order of frequency may be of interest:—

Otitis media	.	.	.	6	13·0 per cent.
Acute broncho-pneumonia	.	.	.	6	13·0 „
Arthritis	.	.	.	4	8·7 „
Acute purulent pericarditis	.	.	.	3	6·5 „
Acute cystitis	.	.	.	3	6·5 „
Mastoiditis	.	.	.	2	4·3 „
Hydrocephalus	.	.	.	2	4·3 „
Chronic phthisis	.	.	.	2	4·3 „

Bedsore	2	4.3 per cent.
Furuncles	2	4.3 "
Septicæmia	2	4.3 "
Acute endocarditis	1	2.2 "
Parotitis (double)	1	2.2 "
Pyonephrosis	1	2.2 "
Sinus thrombosis	1	2.2 "
Cerebral abscess	1	2.2 "

The mortality was 71.7 per cent. These cases occurred before the production of Flexner's serum. COLIN K. RUSSEL.

ACUTE ENCEPHALOMYELITIS. (*Encéphalomyélite aiguë, hémorrhagique, hyperplastique, et diapédétique.*) LAIGNEL-LAVASTINE, *Arch. de Méd. Expér.*, March 1908.

OWING to the extreme differentiation of the elements of the brain, the writer restricts the term "acute encephalitis" to a *particular case* of the cerebral lesions produced by the acute reaction of that organ to the "toxi-infections"; following the phraseology of Metchnikoff, he defines it as "a connective-vascular reaction (*réaction conjonctivo-vasculaire*), a proliferation of the elements of the mesodermic perivascular sheaths, or an infiltration of leucocytes brought into the nervous tissue by diapedesis."

As in the case of other visceral inflammations, there are three types of acute encephalitis, viz., hæmorrhagic, diapedetic, and suppurative. The author aims at showing that there exists a series of transitions between the acute and sub-acute forms, also that inflammatory reactions in the brain and medulla are to be looked on from a similar standpoint.

He analyses minutely a case which presented the clinical picture of an acute ascending paralysis of Landry, and which showed, anatomically, the lesions typical of acute diffuse encephalomyelitis. This *syndrome of Landry* was characterised essentially by a flaccid paralysis, with sensory, vaso-motor, and sphincterial troubles, leading in thirteen days to a quadriplegia, and ending in death four days later.

The lesions found in the brain and spinal cord were very various, and according to the local predominance of such features as hæmorrhages, epithelioid cells, diapedetic perivascular infiltration, or puriform interstitial infiltration, the disease might be called *hæmorrhagic, hyperplastic, diapedetic, or purulent*.

The writer gives a *résumé* of a work already published by Roger Voisin and himself, on the history of the study of acute encephalitis. The first to point out the *rôle* of inflammation in cerebral disease was Broussais, and the idea of encephalitis dates from his time (beginning of nineteenth century). Later, *meningitis*

was clearly differentiated from it, and subsequently also cerebral softening of thrombotic or embolic origin (Virchow). Brissaud and others restricted the term encephalitis to circumscribed cerebral abscess, but it eventually came to include *all the cerebral phenomena observed in the course of the infections and intoxications*. It is only lately in France, however, that encephalitis has been generally recognised as an entity, whereas in Germany a whole series of important works has been devoted to it, beginning with that of Virchow on "The Encephalitis of the New-Born" (1865), and including the epoch-making researches of Wernicke and Strümpell, which established the different types of the disease. The classical German view of encephalitis is summed up in articles by Oppenheim (1897) and Friedmann (1903). Probably the paucity of works on this subject in France as compared with Germany depends partly on a question of words, as many of the cases called encephalitis by the Germans would be reckoned *serous meningitis* in France; this would not hold, however, of the hæmorrhagic form.

After giving full consideration to the various types described by different authorities, the writer comes himself to the conclusion that "acute encephalitis, an inflammatory localisation of the toxoinfections in the brain, includes very complex lesions, and may be grouped, according to the rapidity of its development and the post-mortem conditions, into three categories—hæmorrhagic, diapedetic, and purulent encephalitis." In the first form we have hæmorrhages and red softenings, produced by venous thrombosis of phlebitic origin; there are transition forms between this and diapedetic encephalitis, which is characterised by "the connective-vascular reaction," with various intermediate stages from the passage of a few leucocytes into the perivascular sheaths up to purulent infiltration on the one hand or sclerosis (the sub-acute or hyperplastic form) on the other. Suppuration rarely follows acute encephalitis, though it does occur; it must not be confused with cerebral abscess, of which it is only one type.

To sum up, we have: (1) acute encephalitis—hæmorrhagic, diapedetic, or suppurative; (2) sub-acute—the hyperplastic encephalitis of Hayem; (3) chronic—either so from the first, or as a sequel to the acute or sub-acute forms.

In the case under review the histological findings were exactly the same in brain and medulla; thus the term encephalomyelitis covers the conditions found in these two sections of the nervous system.

The causative agent was undetermined, no bacteria being found. "At most, the multiplicity and variety of the lesions allow one to bring forward the hypothesis of a two-fold process—the first sub-acute, in the course of which the hyperplastic reaction began;

this was surprised in its development by a new and more violent process, which determined the hæmorrhagic and diapedetic form ; on the threshold of suppuration this was arrested by death."

A full bibliography is appended. ARTHUR J. BROCK.

**SOME OF THE COMMONER SYMPTOMS OF CEREBELLAR
(307) ABSCESS.** W. TROTTER, *Brit. Med. Journ.*, March 14, 1908.

THE writer records two cases, the full clinical report of one of which is given, while a short summary is given of the other.

D. K. HENDERSON.

BRONCHIECTASIS AND CEREBRAL ABSCESS. (*Bronchiektasie
(308) und Hirnabszess.*) V. GRÜNBERGER, *Prag. med. Wchn.*,
April 2.

CEREBRAL abscesses coming from purulent or gangrenous processes in the lung cause more marked symptoms than metastatic abscesses originating elsewhere—than those, *e.g.*, occurring in general pyæmia, where the local cerebral condition may be almost hidden by the general systemic disturbance. The writer details the case of a man of thirty, who, during convalescence from pneumonia, began to develop headaches and slight convulsive attacks, along with symptoms of intra-pulmonary suppuration. In hospital the convulsive seizures became more frequent and severe; a clinical diagnosis was made of gangrenous bronchiectatic cavities with metastatic abscesses affecting the right motor area of the brain, thereby producing left-sided hemiplegia and slight hemianæsthesia, with unilateral transient convulsions. Stupor supervened, and patient eventually died. The *sectio* confirmed the diagnosis. Contrary to the usual rule, the cerebral abscess was solitary, and not on the same side as the lung affection; the symptoms, however, pointed typically to a localisation in the motor region of the cortex, and were Jacksonian in character; these irritative phenomena gradually gave place to symptoms of paralysis as the abscess grew in size. Certain marked disturbances of sensibility which appeared in this case are not the rule in pulmonary cerebral abscess. The cerebro-spinal fluid obtained by lumbar puncture was quite clear, and, contrary to expectation, showed no polynuclear cell-elements, but only lymphocytes; here, as often in these cases, had other characteristic symptoms been absent, the cytological finding would have given little, if any, help in diagnosis.

The abscess finally ruptured into the lateral ventricles; despite its superficial situation, the progress of the disease was too rapid to allow of operation.

ARTHUR J. BROCK.

TWO CASES OF CEREBRAL SCLEROSIS OF PSEUDO-BULBAR

(309) **TYPE IN CHILDREN.** ARMAND-DELILLE et GIRY, *Arch. de Méd. des Enfants*, Feb. 1908, p. 126.

A SHORT clinical note of two children of four years with glosso-labio-laryngeal symptoms — dysarthria, dysphagia, salivation, paresis of muscles of palate, tongue and lips. Intellectual deficiency relatively slight. Upper face intact. General attitude like that of a child with Little's disease—limbs contracted and spastic. The cases resemble others recorded by Sicard and Huette, Variot and Roy, by Oppenheim and by Bouchaud. In the two latter autopsy showed atrophy of the lower Rolandic area.

J. H. HARVEY PIRIE.

HYSTERICAL HEMIPLEGIA. (Ueber hysterische Hemiplegie.)

(310) ERNST SCHULZE, *Deut. med. Wochenschrift*, March 26, 1908, p. 544.

THE report of an interesting case of nine years' duration in a man of sixty-five. He had a flaccid paralysis of the right arm and leg, without atrophy. Reflexes normal. Gait typical of a hysterical hemiplegia. Spasm of right genio-glossus, causing protrusion of the tongue to the left. Spasm of the right orbicularis oculi causing a pseudo-paralytic ptosis. Some spastic appearance of the right facial muscles and less marked of the left. Complete right-sided sensory anæsthesia, sharply bounded by the middle line, affecting mucous membranes and testicle as well as skin. Other hysterical stigmata—such as diminution of fields of vision, etc.—Of special interest was the fact that patient mistook right and left sides on both voluntary and passive movement.

J. H. HARVEY PIRIE.

A CASE OF MOTOR APRAXIA, WITH PATHOLOGICAL FIND-

(311) INGS. (Ueber einen Fall von motorischer Apraxie mit Sektionsbefund.) WESTPHAL, *Med. Klinik*, March 1, 1908, p. 283.

THE author describes a case of motor apraxia, complicated, however, by a degree of agnosia. The clinical diagnosis was saturnine encephalopathy, and the apraxic symptoms were highly characteristic, and were much more marked in the left upper extremity than in the right. Perseveration was observed frequently, as well as a certain ideational inertia, a condition which has frequently been remarked in apraxic cases. Post-mortem a considerable degree of internal hydrocephalus was revealed, the left ventricle

being more dilated than the right. The author remarks that macroscopically the corpus callosum appeared normal, but in the present communication no further details are vouchsafed.

S. A. K. WILSON.

CRURAL MONOPLÉGIA, ETC. (*Monoplégie crurale par lésion du* (312) *lobule paracentral, etc.*) LONG, *Nouv. Icon. de la Salpêtrière*, Jan.-Fév. 1908, p. 37.

IN the first case a lesion of the right paracentral lobule occasioned a left crural monoplegia, the arm being unaffected: pathologically the lesion was limited almost entirely to the grey matter, and immediately subjacent white matter, of the ascending frontal convolution on the mesial surface of the hemisphere. The secondary degeneration from this lesion could not be followed in its entirety, but presented the following interesting form: by Weigert's method it was readily detected in the corona radiata, but in the internal capsule, the crus, the pons and the medulla it was not to be seen, while the pyramidal degeneration reappeared in the cord and was traceable as far as the lumbar enlargement. Secondary degeneration was not absent in the areas above noted; but what had happened was that in them there was no interstitial sclerosis to replace the degenerated fibres. Such an absence of neuroglial sclerosis is common in old cases of infantile cerebral hemiplegia, possibly owing to its absorption and to the intermingling of healthy fibres. There seems to be a regional variation of this glial tissue; it does not merely replace degenerated fibres, it is the result of an irritative process of varying intensity.

The author describes a second case, one of congenital dystrophy of the left hand and forearm, with conservation of motility. Death occurred at the age of forty-seven, when it was found that a porencephalic lesion occupied the outer surface of the middle part of the right hemisphere, leaving only the upper third of the Rolandic convolutions. In this case projection fibres from the motor area were readily traceable in the internal capsule, crus, pons, etc. Evidently the fact of the lesion having occurred so early in life rendered compensatory action on the part of the opposite hemisphere more complete, for the defect of motility on the affected side was exceedingly slight. Facial movements were not impaired in any way, although the lower part of the right motor area was non-existent. The interesting feature of the case is the occurrence of projection fibres scattered through the whole of the internal capsule and subjacent pyramidal tract, which suggests supplementary evolution of a descending motor path,

reduced in dimensions, no doubt, but occupying the whole of the usual site of the descending pyramidal path. The author believes that in the course of development the unaffected fibres coming from the upper motor area scattered themselves out through all the internal capsule, etc.

S. A. K. WILSON.

ON FOCAL SYMPTOMS IN DIFFUSE BRAIN DISORDERS.

(313) (*Ueber Herdsymptome bei diffusen Hirnerkrankungen.*) A. SAENGER (of Hamburg), *Münch. med. Wchnschr.*, May 12, 1908.

THE author emphasizes the danger of using focal symptoms for localizing purposes without sufficient allowance for their occurrence in diffuse brain disease. Among the cases of diffuse brain disease in which focal symptoms were in the foreground, he records briefly cases of tuberculous, purulent, sarcomatous, carcinomatous meningitis, of chronic hydrocephalus and cerebral arteriosclerosis; he also refers to the occurrence of focal symptoms in senile brain atrophy and general paralysis.

C. MACFIE CAMPBELL.

CASE OF CEREBELLAR ATROPHY. (*Ueber einen Fall von* (314) *Kleinhirnatrophie.*) STELZNER, *Monatsschr. f. Psychiatr. u. Neur.*, Bd. xxiii., Ht. 3, S. 240, u. Ht. 4, S. 323.

THIS case is described in great detail, including a full account of the microscopical examination. The patient, a woman of thirty-six, showed characteristic cerebellar ataxy, with choreiform tremor, speech disturbances and absent reflexes. Her mother and mother's father suffered similarly. At the autopsy was found a strikingly small cerebellum. The whole cord showed degeneration, with the exception of the anterior and posterior horns and the pyramidal tracts. The differential diagnosis and the various grouping of allied conditions is fully discussed.

ERNEST JONES.

THE MECHANISM OF NYSTAGMUS. Sir W. R. GOWERS. Read (315) at the Neurol. Section of the Roy. Soc. of Med., May 6, 1908.

IT is obvious that nystagmus consists in an alternate contraction of antagonistic muscles, instead of that synchronous action by which the opponents support the acting muscles, while yielding to the movement they produce. Can we discern any similar phenomenon elsewhere? Sherrington's researches on the "Reciprocal Action of Antagonistic Muscles" are of great significance. He found that after division of the cervical spinal cord the lumbar centres pass into a state of automatic action. If a set of muscles

are excited to move a joint, they presently cease to contract, and their stretched opponents contract instead, to cease in turn, and this alternation goes on. He has found that the arrest is due to inhibition of the spinal centre, for it is caused if the nerve to the opponents is divided and the central end stimulated. But this is a purely muscular nerve, hence the inhibition must be due to a stimulus from the stretched opponents, the extension probably acting on the muscle-spindles. The automatic action is thus a reciprocal muscle-reflex process. Symptoms somewhat similar may sometimes be met with in lateral sclerosis. The resemblance of this alternation to nystagmus is close. The observation of structures resembling neural spindles in the ocular muscles by Dr Farquhar Buzzard makes a muscle-reflex process clearer, though the fact that the nerve to each ocular muscle gives a branch to the fifth nerve made afferent impulses from them certain. But nystagmus does not seem to result from a defect of the voluntary impulse; its causes are so situated as to disturb the influences that act directly on the mid-brain reflex and co-ordinating centre for the muscles, which we are compelled to assume. Whether it has a limited position or not, it must be functionally above the motor nuclei, and correspond to the spinal centres for the limbs. It subserves the reflex action of the muscles, including the muscle-reflex action which is here assumed to be, in insubordinate degree, the cause of nystagmus. Other influences must act upon it, including that of light, which is not often conspicuous in adult life, but seems concerned in the causation of miners' nystagmus. A disproportion in the amount of light, excess or defect, during early infancy, seems to determine the alternate action of the opposing muscles in many cases of infantile nystagmus, an indication of the readiness with which the centre may be deranged and of the existence in it of structural arrangements for the muscle-reflex alternation.

The stability of the centre, and its control by the voluntary impulse, seem to depend on the equilibrium of the subsidiary influences that act on it. If any of these are deranged, the muscle-reflex activity asserts itself in the alternate action of nystagmus; it may be only when action is excited by the will, when it is more rapid in the direction of voluntary movement. The derangement of the centre may be partial, and vary much in degree, while it often seems to increase with constant activity. An instance of its production by equilibrial disturbance is the brief nystagmus from labyrinthine causes, such as the impressions excited by rotation of the body. It is probably thus that it results from lesions of the cerebellum. It scarcely ever results from disease of the cerebral hemisphere, and the lesions that give rise to it are seldom precise in their indications, insular sclerosis being

multiple and tumours causing distant pressure. The cause is generally in the mid-brain, pons, or cerebellum.

Striking evidence of the insubordination of the muscle-reflex centre is afforded by the degree to which nystagmus and the quicker motion may attain. The latter is present even in labyrinthine nystagmus. In other forms the alternate action may persist even in the mid-position, and a dominant motion, say quick to the right, may persist even when the eyes are moved to the left, as well as in upward and downward movements. If downward nystagmus preponderates, it may deflect downwards that which should be horizontal. These variations in excess make it less difficult to understand the slight and irregular forms often seen, when the tendency to the muscle-reflex alternation must be only trifling and partial in the centre. The explanation also applies to the form met with in miners' nystagmus, whatever view is taken of its causation.

The complex character of the centre, and the numerous influences by which it is maintained in adjustment, explain the readiness with which it may escape control. Insubordinate activity, once established, tends to increase. It is right to add that Wilbrand's explanation of nystagmus assumes a disorder of the ordinary reflex centre for the eyeball movements and a want of harmony between its condition and that of the volitional impulse. The muscle-reflex action in the centre, which the author believes to be the essential cause of nystagmus, seems to have escaped recognition.

AUTHOR'S ABSTRACT.

PARALYSIS OF UPWARD ASSOCIATED OCULAR MOVEMENTS. SPILLER, *Arch. a. d. Neurol. Instit. a. d. Wien. Univ.*, Bd. xv., T. 1, 1907, p. 352.

THIS and the previous paper by the author published in the *Journ. of Nerv. and Ment. Dis.*, July and August 1905, contain references to the majority of the recorded cases of paralysis of the upward associated movements of the eyes. (One by the abstractor, published in the *Trans. Med.-Chir. Soc. Edin.*, vol. xix., with an abstract and illustrations in the *Rev. Neurol. and Psychiat.*, 1903, p. 441, has not come under the writer's notice.) Persistent palsy of the associated upward and downward movements indicates a lesion near the aqueduct of Sylvius, or, more accurately, near the oculo-motor nuclei. Out of fifty-nine cases of lesion of the corpora quadrigemina, with autopsy, Tödter found such paralysis in nineteen only. Obviously, therefore, proximity of the oculo-motor nucleus to a lesion in the corpora quadrigemina is essential to the production of this symptom. In the abstractor's case there was an

angio-glioma involving the peri-Sylvian grey matter, which had infiltrated the oculo-motor nucleus.

This paper should be read in conjunction with that previously published in the *Journ. of Nerv. and Ment. Dis.*

ALEXANDER BRUCE.

**THE DIAGNOSTIC VALUE OF PUPILLARY IMMOBILITY AND
(317) SLUGGISHNESS IN NERVOUS AND MENTAL DISEASES.**

(Die diagnostische Bedeutung der Pupillenstarre und der Pupillenträgheit für die Erkennung von Nerven- und Geisteskrankheiten.) K. RETZLAFF, Diss., Berlin, 1907, pp. 42.

IMMOBILITY of the pupil, especially reflex immobility to light, occurs in general paralysis in about 50 per cent., in tabes in about 80 per cent. of all cases; it is an early symptom, and may, as an isolated phenomenon, precede the outbreak of these disorders ten and more years. The work deserves mention chiefly on account of the bibliographical index, with 265 references.

F. LOEB (C.g.B.).

CONTRIBUTION TO THE THEORY OF FACIAL PARALYSIS.

(318) (Beitrag zur Lehre von der Facialislähmung.) BERNHARDT, *Monatsschr. f. Psychiatr. u. Neur.*, März 1908, S. 191.

A POLEMIC aroused by Lipschitz's recent work on the subject. Bernhardt defends his view that fibrillary tremor is due to excitability of the facial nucleus. He further maintains that Lipschitz's work on the irregular distribution of regenerating fibres can be brought fully into harmony with Bethe's hypothesis of partially autogenous regeneration of nerves.

ERNEST JONES.

ON TORTICOLLIS. (A propos de torticollis.) BIENFAIT, *Journ. de*
(319) *Neur.*, Feb. 5, 1908, p. 141.

THE paper concerns a case of "idiopathic" spasmodic torticollis, and does not contain any feature of interest beyond discursive remarks on the atrophy of the uninvolved sternomastoid.

S. A. K. WILSON.

**NOTES ON A CASE OF GRAVES' DISEASE TREATED BY
(320) THYROIDECTOMY.** TAYLOR, *Med. Press*, April 15, 1908.

THE case was that of a dressmaker aged 24 years, who had been suffering from the condition for over five years, and

exhibited characteristic symptoms of a severe type. Half an hour before operation $\frac{1}{2}$ gr. morphine and $\frac{1}{100}$ gr. atrophine were given hypodermically. Chloroform was the anæsthetic administered. The greatly enlarged right lobe and almost the whole of the isthmus were removed. From the second day onwards the pulse gradually fell in frequency, and on the tenth day was about 80 per minute; prior to the operation it had varied from 120 to 136. The operation was performed on May 7, 1906. The eyes are still prominent, but much less so than formerly. The palpitation, muscular tremors, and nervousness are now quite gone. The writer discusses the various surgical procedures adopted in the treatment of exophthalmic goitre, and indicates that the best authorities recommend thyroidectomy in preference to division or removal of the isthmus, sympathectomy, or ligature of two or more of the thyroid arteries. When symptoms of Graves' disease have developed secondarily in patients who have been the subjects of an ordinary colloid goitre for some considerable time, the mortality following operation is lower and the prospects of permanent cure better. In conclusion, more frequent and earlier recourse to surgical treatment is advocated.

HENRY J. DUNBAR.

EXOPHTHALMIC GOITRE. (*Morbus Basedowii*.) L. v. SCHRÖTTER, (321) *Med. Klinik*, 5th April.

THIS article, based on a clinical lecture, begins with a demonstration of the characteristic symptoms of the disease. Special attention is given to the occurrence in one case of an excessive accumulation of subcutaneous fat, confined to the hypogastrium and lower extremities. The results of the analysis of this fat are given; it did not differ in composition from that of the skin of other parts. The occurrence of lipomatosis in this case was noteworthy in view of the loss of weight and disappearance of fatty tissue which is so usual in exophthalmic goitre.

Morbus Basedowii is commonly looked on as the antithesis of myxœdema, the one depending on defective, the other on excessive functioning of the thyroid. Setting aside, however, the puzzling occurrence of cases of exophthalmic goitre without thyroid enlargement, and tumour of the thyroid without exophthalmic goitre, this theory does not explain whether the secretion of the gland is added to the fluids of the body in order to maintain some special function or to render certain other fluids innocuous. In view of the varying size of the gland in different cases, it is probable that the quality as well as the quantity of the secretion is of importance; and while typical Basedow's disease may

be designated *hyperthyroidism*, and myxœdema and its allies *athyroidism*, there is yet another form, *dysthyroidism*, which includes the absolutely atypical cases. The fact that among these we may get pigmentary skin affections, striking depositions of fat, etc., suggests that several of the organs which preside over internal secretion may be affected simultaneously.

ARTHUR J. BROCK.

RESPIRATORY CHANGES OF CENTRAL ORIGIN. (Über einige (322) Respirationsveränderungen centralen Ursprungs.) FRUGONI, *Neur. Centralbl.*, March 1, 1908, p. 202.

GROCCO has described a form of dissociated respiration in which the synergy of thorax and diaphragm is so altered that one is in the inspiratory phase while the other is in the expiratory. This dissociated respiration is to be seen in meningitis, cerebral tumour and abscess, in general diseases of various kinds—in fact, in any case of functional disturbance of bulbar centres from toxæmia, infection, asphyxia, etc. The author has observed a similar phenomenon in deep chloroform narcosis, on the near approach of a state of collapse. Tracings are given from a case of cerebro-spinal meningitis, in which *sub finem* it was observed that while the thorax was expanding and falling more or less regularly the diaphragm was in a state of “hallorhythm”: it was contracting spasmodically, every third contraction being much stronger than the others, and producing a sobbing type of respiration; the phases of the thoracic and the diaphragmatic musculature no longer corresponded. This clonic “hallorhythmic” spasm of the diaphragm is occasioned probably by a “hallorhythmic” discharge from the bulbar centres to the cells of origin of the phrenic nerve in the spinal cord.

S. A. K. WILSON.

A NEW CASE OF CHRONIC TROPHŒDEMA. (Sur un nouveau cas (323) de trophœdème chronique, etc.) PARHON and CAZACON, *Nouv. Icon. de la Salpêtrière*, Nov.-Dec. 1907, p. 448.

A YOUNG woman, 35 years old, had noticed for ten years a slowly increasing symmetrical swelling of the legs and thighs, without any change in the colour of the skin. During the same time she complained of apathy, debility, a degree of agoraphobia, headache, involuntary quivering of the muscles, and a feeling as of falling backward. On examination her urine was normal. Her legs presented a firm elastic pseudo-œdema, which was painless on pressure and was but slightly dimpled by the application of the finger. This œdema extended from the groins to the malleoli.

The etiology and pathogenesis of trophœdema are still obscure.

Its segmental topography suggests a medullary origin. Valobra (*cf. Review of Neurology and Psychiatry*, 1905, p. 628) has pointed out the relation between urticaria, Quinke's circumscribed œdema, and trophœdema, attributing all to a disturbance of lymph secretion. In urticaria, acute lymphatic œdema is associated with vasomotor phenomena; in trophœdema there is secondary hyperplasia of subcutaneous connective tissue. But there may be a form of trophœdema which is independent of the action of the nervous system, if Hertoghe's views of its dependence on hypothyroidism be correct. Wright believes that urticaria is a consequence of a diminution in the coagulability of the blood, and considers urticarias and acute œdemas to be serous hæmorrhages. Hence there may be a connection between chronic trophœdema and the metabolism of the lime salts of the body. The thyroid gland plays an important rôle in the assimilation of calcium. This defect in metabolism facilitates the transudation of lymph, which, if long continued, leads to the formation of fatty connective tissue, as in trophœdema, adenolipomatosis, myxœdematous infiltration, etc.

S. A. K. WILSON.

A NEW DIAGNOSTIC SIGN IN RECURRENT LARYNGEAL (324) PARALYSIS. A. R. ALLEN, M.D., *Journ. Nerv. and Ment. Dis.*, March 1908.

THE author has noticed that in cases of paralysis of one recurrent laryngeal nerve there is a very material difference in the upward excursion of pitch when the vocal apparatus is stimulated electrically during the singing of a tone. A small button electrode is placed over the lateral part of the crico-thyroid membrane, and the patient is instructed to sing the note C: for a man the note is an octave lower than for a woman, and for both the notes fixed on are free from muscular strain. On the normal side there will be a rise in pitch equal to from seven to fourteen half-tones, whilst on the paralysed side the note will only be raised from two to three half tones. Allen says that this test permits a quantitative estimation of the contractile ability remaining in a vocal cord.

J. S. FRASER.

THE CORPUSCULAR RESISTANCE AND THE HÆMOLYTIC (325) POWER OF THE SERUM OF EPILEPTICS. (*La résistance globulaire et le pouvoir hémolytique du sérum chez les épileptiques.*) H. CLAUDE, A. SCHMIERGELD, and A. BLANCHETIÈRE, *L'Encéphale*, No. 3, 1908.

THE authors repeated a series of experiments on the blood of epileptics, similar to those made by de Buck last year, but except on one or two minor points were unable to confirm his results.

They found, as did de Buck, that the corpuscular resistance of the non-heated blood of epileptics is normal or sub-normal, and with perhaps a slight diminution during active periods of the illness.

The corpuscular resistance after heating is generally strongly diminished among epileptics, but so it is in exactly the same manner in other maladies, in normal persons, and in animals. Therefore this observation is of no diagnostic significance. The amount of water necessary to be added to the serum to excite hæmolysis is not less in epileptics than in normal persons. After heating the serum (to 56° C. for a quarter to half an hour) the escape of the hæmoglobin does not vary, or varies in very slight degrees. This variation is quite as marked with the serum of non-epileptics as with that of epileptics.

They therefore conclude that the examination of the corpuscular resistance, or of the hæmolytic power of the serum, does not supply any new means of diagnosing epilepsy.

JOHN TURNER.

CHRONIC ALCOHOLISM IN A CHILD. (*Alcoolisme chronique (326) chez un enfant.*) BOULENGER, *Journ. Neurol.*, Feb. 5, 1908.

THIS is an account of a boy, aged nine, with a strong alcoholic family history. With the concurrence of his parents, he was in the habit of drinking daily large quantities of beer; and he was familiar with the taste of many liqueurs and other alcoholic beverages.

There was hypertrophy of the liver and thickening of the radial arteries. Tremor was induced by attempting any fine movement, such as writing or drawing. The boy suffered from visual hallucinations, morning headache, feebleness of attention, loss of memory, and general backwardness. Euphoria and logorhoea were well marked.

W. H. B. STODDART.

HEADACHES CAUSED BY PATHOLOGIC CONDITIONS OF THE (327) NOSE AND ITS ACCESSORY SINUSES. GERHARD H. COCKS, M.D., and JOHN E. MACKENTY, M.D., *Arch. of Otol.*, February 1908.

ACCORDING to the authors, headache of nasal origin may be classified under two heads:—(1) non-inflammatory, due to enlargement of the middle turbinal, adhesions of the middle or inferior turbinal to the septum nasi, deviation of the septum pressing on the turbinals, or combinations of these conditions; and (2) inflammatory conditions—which again may be due to either chronic or

acute sinusitis. Persons constitutionally prone to headaches (neuropathic temperament) are more liable to suffer than normal individuals. Three cases are given in which the sub-mucous resection of the deviated nasal septum resulted in the cure of severe headache of long standing. Case 4, in which the headache was stated to be due to pressure of the middle turbinals on the septum, was less successful: the patient was neurotic, and the writers intend to perform a further operation. The following three cases deal with chronic inflammation of the ethmoidal labyrinth and maxillary antra: headache, which was present before operation, was either cured or markedly relieved. Three cases of acute suppuration in the maxillary antrum, frontal sinus, or in both cavities combined, conclude the series: treatment resulted in cure of headache in all three. The situation of the pain in the head associated with suppuration in the various sinuses, as stated by Cocks and MacKenty, differs considerably from that found in most of the text-books, *e.g.* "In maxillary sinusitis the usual point for the pain is over the anterior surface of the antrum." Lack says that in acute cases the pain is situated in the infra-orbital region, over the malar bone, and in the teeth of the upper jaw. Killian, Hartmann, and Hajek state that pain may occur in the supra-orbital region. The marked periodicity of the pain in frontal sinus suppuration is very characteristic—usually beginning about 10 A.M. and going on till 2 P.M. or 4 P.M., when it suddenly ceases. In sphenoidal sinus suppuration the headache is in the occipital region. Finally the authors give a short account of the nerve supply of the nose and its accessory sinuses in order to explain the situation of the headache: as they themselves admit, a good deal of work has still to be done on this subject.

J. S. FRASER.

PSYCHIATRY.

COMPLEXES AND THE ÆTIOLOGICAL FACTORS IN DEMENTIA

(328) **PRECOX.** (*Komplexe und Krankheitsursachen bei Dementia praecox.*) BLEULER and JUNG, *Zentralbl. f. Psychiatr. u. Nervenheilk.*, 1908, Nr. 257, S. 220.

THIS is an article of Bleuler's written mainly to define his attitude towards Jung's work on dementia praecox, in reply to one by Meyer in which several misunderstandings occur. Bleuler is very precise in his views, which shortly are as follows: Some as yet unknown organic process underlies every case of dementia praecox and is the essential cause of the disease-process; probably some of the psychical symptoms (called primary) are the direct result of this

process ; by far the majority of the symptoms (called secondary), however, are determined by the operation of various feeling-infused complexes acting on the pathological basis just mentioned. Thus the disease would clinically be to a large extent *latent* were it not for the action of these complexes, which renders it *manifest* by creating the familiar symptomatology of delusions, hallucinations, obstruction, etc. He gives a number of throughout convincing reasons for the second view, though the first view concerning the organic process is given only as an opinion.

In a short addendum Jung criticises Bleuler's article. He agrees with all the main points, and differs only in leaving the cause of the organic predisposition—which he fully accepts—more open, even suggesting that it may occasionally be produced by a primary affective process.

ERNEST JONES.

IDIOCY AND DEMENTIA PRÆCOX. (*Idiotie und Dementia* (329) *præcox*.) W. WEYGANDT (of Würzburg), *Zeitschr. f. d. Erforsch. u. Behandl. d. Jugend. Schwachs.*, 1906, Bd. 1, p. 311.

THE author discusses the relationship of idiocy to dementia præcox on the basis of illustrative cases, and comes to the following conclusions.

1. Many cases of dementia præcox have already shown in childhood peculiar traits, without the presence of idiocy or even of mild dementia.

2. Frequently one observes the outbreak of one of the three main forms of dementia præcox in imbeciles whose mental enfeeblement had previously shown no characteristic symptom of dementia præcox.

3. Idiots with deterioration of the apperceptive and emotional life are not to be considered as allied to cases of dementia præcox when other ætiological factors are obviously at work.

4. Numerous motor disorders in idiots, which are similar to those met with in dementia præcox, are found in cases of the most varied ætiology. These symptoms are not to be regarded as pointing to dementia præcox, but are to be explained in the same way as analogous symptoms in catatonics ; indications of these phenomena on the basis of still inco-ordinated motor impulses are to be seen in normal evolution at a certain youthful period.

5. There are cases where during childhood, after a series of normal years, a dementia sets in, which resembles dementia præcox in many features without the resemblance being complete ; these cases may be called dementia infantilis ; their explanation is quite obscure.

C. MACFIE CAMPBELL.

FORCED SPEECH IN MANIC-DEPRESSIVE INSANITY. (Der

(330) *Rededrang im manisch-depressiven Irresein—Spez. die dialogisierende Manie.*) PFERSDORFF, *Zentralbl. f. Psychiatr. u. Nervenheilk.*, 1908, Nr. 257, S. 209.

A VALUABLE casuistic article. Four cases are described, only the forced speech (*Rededrang*), not the results of communicative impulse (*Mittheilungsdrang*), being given. The associations found are discussed, and attention called to the frequent impulse to translate and to spell answers to a question the patient puts to himself. The main thesis of the article is that manic-depressive speech can be divided into a motor variety (characterised by word-stem associations) and a sensory (characterised by sound associations).

ERNEST JONES.

SOME REMARKS UPON THE TERM MANIC-DEPRESSIVE

(331) **INSANITY.** (*Einige Worte betreffs der Benennung "manisch-depressives Irresein."*) A. WIZEL, *Neurol. Centralbl.*, April 16, 1908, p. 368.

IN this short paper Wizel criticises the term introduced by Kraepelin to designate one of his groups of mental disease. Wizel points out that depression is a term which has long been used to indicate a well-recognised mental symptom, while melancholia has just as clearly been regarded as expressive of a disease. He considers that this usage should be strictly adhered to, and that consequently Kraepelin's term should be altered to "manic-melancholic insanity." Strictly speaking there appear to be good grounds for this suggestion.

JAS. MIDDLEMASS.

CRANIAL TRAUMATISM AND MENTAL DISORDER. (Trau-

(332) *matismes Craniens et Troubles Mentaux.*) ROGER DUPOUY and RENÉ CHARPENTIER, *L'Encéphale*, April 1908.

SINCE the passing of the French law of 1898 relating to compensation for accidents to employees, increasing interest has attached to the connection between head injury and insanity, what at one time had a purely ætiological value having now medico-legal importance. Large numbers of cases have been investigated, and the general trend of opinion appears to be in favour of the belief that, except in rare instances, head injury is not a cause *per se* of insanity, but in nearly every case merely an exciting factor. With this opinion Drs Dupouy and Charpentier are in agreement, and the present paper is a careful and minute description of a single and very

interesting case of traumatic insanity terminating in recovery. Before discussing the case the authors outline the development—mainly in France—of alienist thought on this matter from the time of Pinel and Esquirol up to to-day, and the effect of the law of 1898 in producing what Prof. Brissand has baptised recently under the name of *sinistrosis*, this resulting from an *idée fixe* that every accident in the course of labour constitutes a damage to be followed by compensation. They recall, from the remarkable *Études expérimentales sur les traumatismes cérébraux* of Duret, the statement that “if the lesion (traumatic) be slight and consist of a small effusion of blood or of a zone of vascular irritation, the functions of the centres concerned are exalted; if the lesion be destructive the functions are abolished,” and accepting a strict parallel between mental processes and the nervous processes with which Duret’s experiments were concerned, the authors advance the following propositions:—

1. The traumatism reveals the special inherent predisposition of the individual suffering the head injury. The more marked the predisposition the less the injury needed to determine the same mental disorders.
2. Head trauma may disclose hysterical or epileptic phenomena in a potential hysteric or epileptic, delirium tremens in a chronic alcoholic, or mental confusion in the subject of auto-intoxication (hepatic, renal, etc.).
3. Slight lesions determine exaltation of the intellectual functions; destructive lesions abolition of these functions.
4. Post-traumatic dementia is always the result of profound and extensive cerebral lesions. This form of dementia may, in predisposed individuals, simulate true general paralysis.

The case the authors describe responds to the statement of Duret, that after slight lesions the functions are exalted, and, further, that the traumatism reveals the special inherent predisposition. The case was that of a man of 38 years who was knocked down by a cyclist, sustaining a blow upon the head, followed by unconsciousness and bleeding from the nose and mouth. He remained in a semi-comatose condition for three days, and on the fourth recovered consciousness, but was agitated, restless, and abusive to his wife, continually laughing and making absurd statements, euphoric, disoriented in both space and time, markedly amnesic (retro-antegrade amnesia), paramnesic, and incoherent in speech, with confabulation and auditory hallucinations. He had transitory and alternate irregularity of the pupils, which were otherwise normal, hand tremor, increased superficial and deep reflexes, and general hyperæsthesia. The accident

occurred on September 22, 1907, and his mental condition remained practically unchanged till the end of October, after which he steadily improved, and was discharged from the asylum of St Anne recovered, with only slight euphoria remaining. The patient had been an alcoholic, and some of his symptoms were due, in the authors' opinion, to this former alcoholism. He had had typhoid fever, followed by hepatic insufficiency (indicated by slight jaundice, pruritis, hypoazoturia and indicanuria), the marked mental confusion being due to trauma in the subject of auto-intoxication, in harmony with their statement 2. Examination of the cerebro-spinal fluid at the beginning of the illness showed that there had been some subarachnoid hæmorrhage, whilst eight weeks later lumbar puncture gave negative results.

R. CUNYNGHAM BROWN.

CHRONIC PSYCHOPOLYNEURITIS. DUPRÉ and CHARPENTIER, (333) *L'Encephale*, April 1908, p. 289.

It is instructive to analyse cases of Korsakoff's syndrome in which cure has been incomplete, for such cases present a combination of the physical symptoms of chronic polyneuritis with a peculiar mental enfeeblement, characterised by defect of memory, incapacity for retaining impressions, disorientation, chronic confusion and impairment of work-a-day activity, such as to render the patient quite incapable of doing anything for himself. A case of this description is narrated by the authors, where a woman presented these symptoms in characteristic manner ten years after an attack of influenza complicated by alcoholism.

S. A. K. WILSON.

ON IDIOCY SECONDARY TO DISEASE OF THE CEREBRAL
(334) **VESSELS.** (Ueber eine zu "Idiotie" führende Erkrankung (Angiodystrophia cerebri.) O. RANKE (of Wiesloch), *Zeitschr. f. d. Erforsch. u. Behandl. d. Jugend. Schwachs.*, 1907, Bd. i., p. 122.

THE clinical and anatomical record of the case of a 9½ year old idiot, without gross paralysis or epileptic phenomena. Histologically the nerve-cells of the cortex presented marked degenerative changes, the glia showed proliferation with marked regressive changes; the cerebral vessels were profoundly altered, being to a large extent replaced by rigid, highly refracting tubes with complete absence of nuclei.

The histological picture was that of a primary vascular disease with secondary parenchymatous and interstitial changes.

C. MACFIE CAMPBELL.

THE VICISSITUDES OF A CASE OF MENTAL TORTICOLLIS.

(335) (*Les péripéties d'un torticollis mental.*) MEIGE, *Nouv. Icon. de la Salpêtrière*, Nov-Dec. 1907, p. 461.

THIS is a long and detailed account of a case of mental torticollis observed for a period of six years, in which a perfect cure has resulted from treatment by exercises of various kinds. The description of the case is interesting, and well merits careful reading in the original. At one period in the history the symptoms were so aggravated as to appear hopeless to any one with less confidence in his methods than M. Meige, but in the end the patient was restored to normal health, and he has continued absolutely well for two years (since January 1906).

S. A. K. WILSON.

THE MENTAL TORTICOLLIS OF BRISSAUD: FAILURE OF

(336) **SURGICAL TREATMENT.** (*Torticollis mental du Brissaud: insuccès du traitement chirurgical.*) SICARD and DESCOMPS, *Nouv. Icon. de la Salpêtrière*, Nov-Dec. 1907, p. 459.

It may be remarked that the title of this brief paper is misleading, inasmuch as the surgical treatment adopted for this case of retrocollis, viz., division of the *muscles* trapezius, splenius, complexus and inferior oblique, on both sides, is a form of surgical interference which is admittedly unsatisfactory. The remarks of the authors on the futility of surgical procedure are rather uncalled for, since no attempt was made to treat the patient by division of the posterior primary divisions of the upper cervical roots.

S. A. K. WILSON.

GROUPING OF HOMOSEXUALS. (Einteilung der Homosexuellen.)

(337) NÄCKE, *Allg. Zeitschr. f. Psychiatr.*, Bd. lxxv., Ht. 1, S. 109.

AFTER some preliminary remarks on the unrecognised frequency of this affection, Näcke discusses the definition of it and the modes whereby it may be grouped. He just divides the cases into pure homosexuals and bisexuals, and then adopts a chronological classification into the childhood, puberty and adult life cases, according to the time at which the perversion becomes manifest; of the three last, the second is by far the largest group.

ERNEST JONES.

DIAGNOSIS OF HOMOSEXUALITY. (Die Diagnose der Homo-
(338) sexualität.) NÄCKE, *Neurolog. Centralbl.*, April 16, 1908,
S. 338.

NÄCKE here relates the principal characteristic points of this perversion, the diagnosis of which is often so difficult. He lays especial stress on the study of the dreams of doubtful cases.

ERNEST JONES.

SIMULATION OF INSANITY. (Zur Frage der Simulation von
(339) Geisteskrankheit.) RIEHM, *Allg. Zeitschr. f. Psychiatr.*, Bd.
LXV., Ht. 1, S. 28.

THIS article, 80 pages strong, does not lend itself to abstracting. It consists almost exclusively in an account of two interesting cases, which are described at great length.

ERNEST JONES.

**THE COÖPERATION OF THE ALIENIST IN THE CARE AND
(340) TRAINING OF DEFECTIVES.** (Die Mitwirkung des
Psychiaters bei der Fürsorgeerziehung.) O. KLUGE (of Pots-
dam), *Zeitschr. f. d. Erforsch. u. Behandl. d. Jugend. Schwachs.*,
1907, Bd. i., p. 311.

KLUGE discusses the social and pedagogical aspects of the question of dealing with defectives, and at the end of his communication he formulates in outline the principles which should guide those responsible for carrying out reforms.

Among those who require care and special training are cases with all degrees of mental enfeeblement up to complete idiocy, and of mental disorder up to well-defined insanity. Such children should be examined as soon as possible by an experienced alienist, and their future training should be under similar supervision. For efficient treatment the children should be separated according to their age, degree of mental impairment, and degree of educability. As to the nature of the institutions suitable for the different classes, and the advantages of a central bureau, the author goes into some detail.

C. MACFIE CAMPBELL.

Reviews

THE MAJOR SYMPTOMS OF HYSTERIA. Fifteen Lectures given in the Medical School of Harvard University. By PIERRE JANET, M.D. New York: The Macmillan Company, 1907. Price 7s 6d net.

IN this series of lectures, delivered in the Harvard Medical School in October and November 1906, Professor Janet presents the subject of hysteria with the same lucidity and charm of style which characterise his French works. It is seldom that one has the pleasure of reading in English a scientific book which combines to such a degree accuracy of expression with simplicity and vividness of phrase.

The author begins by discussing somnambulism, "the most typical, the most characteristic symptom of hysteria." Illustrative examples are given of the simplest type of somnambulism, where the patient is absorbed with the enacting of some emotional situation. Here the system of ideas relative to an event has emancipated itself, and develops on its own account, and to an exaggerated degree.

From such monoideic somnambulisms it is but a step to fugues and polyideic somnambulisms and conditions of double personality, where a much more complex group of systems, of thoughts and feelings, becomes dissociated and develops in an independent manner. In these conditions the dissociation of a psychological system is associated with amnesia, not only of the somnambulatory episode, but occasionally of the emotional event leading to the dissociation.

As systems of thought become dissociated, so may functional systems of movements, and these may show an analogous independent and exaggerated development, giving rise to tics and choreas on the one hand, and on the other hand to paralyses and anæsthesias which play the same rôle as the amnesias of somnambulism.

In all these phenomena functions are not destroyed but only suppressed, no longer at the disposal of the will and consciousness of the subject, but dissociated from that congeries of psychological systems which is called the personality. Just as on the motor side are observed contractures and paralyses, tics and choreas due to the abnormal functioning of a psychological system which has attained some independence, so on the sensory side groups of sensations become split off and attain a certain independence, and thus we have tics, pains, hallucinations on the one hand and

various anæsthesias on the other. These remarks apply not only to the more complex and highly elaborated functions at play in the adaptation of the individual in everyday life, they apply also to the anorexias, vomitings, dyspnœas where there is an analogous emancipation of the cerebral and psychological functions relative to the visceral organs. This emancipation leads on the one hand to an exaggeration of the function, on the other to a disappearance from consciousness of certain organic wants and reactions.

Having devoted one lecture to each of the following subjects, Monoideic Somnambulisms, Fugues and Polyideic Somnambulisms, Double Personalities, Convulsive Attacks—Fits of Sleep—Artificial Somnambulisms, Motor Agitations—Contractures, Paralyzes, Professor Janet devotes his eighth lecture to the Psychological Conception of Paralyzes and Anæsthesias. In this lecture he demonstrates the essential unity of the mechanism in the above disorders, and shows how certain systematic paralyzes are due to the dissociation and abnormal independence of certain systems of movements grouped by education; these paralyzes are the result of psychological dissociation, which is likewise at the basis of hysterical choreas and tics.

The following four lectures discuss the Troubles of Vision, the Troubles of Speech, the Disturbances of Alimentation, Tics of Respiration and Alimentation. Having thus presented the major symptoms of the disorder, Professor Janet devotes two lectures to an analysis of the mental status of his patients in order to discover the fundamental stigmata of the hysterical. He concludes that the most important mental stigma of hysteria is suggestibility. The term suggestion has been rather loosely applied in the discussion of hysteria, and Janet takes considerable pains to give it a precise meaning. Suggestibility is not exaggerated docility, it requires for its occurrence two conditions—the preservation of automatism and the diminution of personal synthesis; suggestion designates “a very special fact, the complete development of an idea which takes place without the will and the personal perception of the subject”; it implies a malady of the personality, a diminution of personal synthesis. A second stigma of equal importance is a characteristic “disposition to indifference, to abstraction, to quite exaggerated absent-mindedness.” What is not in the field of attention is for these individuals non-existent; there is no penumbra round the central field of attention. To a certain extent it is this very absence of indistinct, but ever present and controlling, shadowy background which permits of the uncontrolled development of ideas, *i.e.* of suggestion, in the hysterical. A third stigma is the phenomenon of transfers and equivalences, the replacement of one accident by another, which it would be a mistake to consider as always due to suggestion. Professor Janet

brings these three stigmata—suggestion, absent-mindedness and alternation—under the one conception of “retraction of the field of consciousness.”

Besides these stigmata the hysterical have symptoms common to the large group of neuropathic individuals, and depending upon a lowering of the higher functions of the mind. The essential stigma of hysteria, therefore, is a lowering of the mental level, which takes the special form of a retraction of the field of consciousness. This lowering of the mental level is specially apt to occur at certain physiological periods, *e.g.* puberty, after exhausting diseases, after the strain of emotion.

In the last chapter Professor Janet reviews the typical symptoms of hysteria and the definitions of it, which have been attempted, and enunciates five laws in which he sums up his conception of the disorder. The fifth law is thus expressed: “We remark a very curious fact, which we recognise without always being able to account for it. The dissociation bears on the function that was in full activity at the moment of a great emotion.”

This law is of such fundamental importance in the pathogenesis of hysteria that further elaboration along this line would have been extremely welcome, but in these lectures Professor Janet has confined himself strictly to a symptomatic analysis of hysteria. There is no more finished study of a complex mental disorder than this series of fifteen lectures.

C. MACFIE CAMPBELL.

BISMARCK IM LICHT DER NATURWISSENSCHAFT. Von
GEORG LOMER. Halle a. S., Carl Marhold, 1907, pp. 160.
Price, 3 M.

IN this book the author presents to the reader Bismarck's personality from the “point of view of anthropology, psychology, and medicine.” Political events are referred to only in so far as is necessary for the understanding of Bismarck's development. Behind the man of steel and iron we find the man of highly sensitive and sometimes overstrung nature, who at a comparatively early age began to suffer from various disorders, largely nervous in origin. After passing the sixties these disorders became more marked and periods of nervous irritability were frequent. In his life one traces a certain rhythm, periods of successful accomplishment and harmonious activity alternating with periods of depression and lack of self-confidence.

His habits were in many points contrary to the rules of hygiene, but the author vigorously repudiates the view that he

was a chronic alcoholic or morphine habitué, although in later life he frequently made use of opiates.

The author never digresses from the main theme, notwithstanding numerous temptations to do so, and has given in comparatively short compass an extremely interesting and well-reasoned account of an outstanding personality.

C. MACFIE CAMPBELL.

**EXPOSÉ DES TITRES ET TRAVAUX SCIENTIFIQUES DU
DOCTEUR PIERRE MARIE.** Paris: Masson et Cie, 1908.

THIS collection of Professor Pierre Marie's scientific works will be welcomed by every neurologist. It is in itself a large volume of 247 beautifully printed and illustrated pages, and yet it contains merely the references to or short abstracts of the work of this great clinician. It will be of the utmost assistance as a guide to the source of his original papers.

ALEXANDER BRUCE.

UNIVERSITY OF PENNSYLVANIA. Contributions from the Department of Neurology and the Laboratory of Neuropathology for the year 1906. Vol. ii., Philadelphia.

WE again acknowledge receipt of the second volume of this valuable collection of reprints from the Department of Neurology of the University of Pennsylvania. It does not call for detailed review, as most of the papers have been already abstracted in the *Review*, but it forms a remarkable tribute to the energy and scientific zeal of the Philadelphia School.

ALEXANDER BRUCE.

ARCHIV FÜR GESCHICHTE DER MEDIZIN. Herausgegeben von der Puschmann-Stiftung an der Universität Leipzig unter Redaktion von KARL SUDHOFF. Bd. i., Heft 1. Mit sieben Abbildungen. Leipzig: J. A. Barth, 1907.

THE name of the editor of this new Archiv is a sufficient guarantee of the serious nature of the undertaking. That there is room for such a journal no one will deny, and it will be much welcomed by those who, while taking an interest in the history of medicine, have insufficient time or lack of the necessary training to indulge in these somewhat recondite studies. The new journal is not intended to be a mere review or critical organ, but rather to be

an organ for the publication of original work in this branch, and the original contributions may appear in any of the four chief European languages. To judge from the variety of the communications in the first three numbers and the solid contribution which they make to the subjects treated, this Archiv will be a valuable addition to contemporary periodical literature.

C. MACFIE CAMPBELL.

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Ludwig Edinger. "Vorlesungen über den Bau der nervösen Zentralorgane des Menschen und der Tiere." Zweiter Band. Siebente, ungewerkelte Auflage. Vogel, Leipzig, 1908, M. 15.

Review

of

Neurology and Psychiatry

Original Articles

A CASE OF DISEASE OF THE POST-CENTRAL GYRUS ASSOCIATED WITH ASTEREOGNOSIS.

By PURVES STEWART, M.A., M.D., F.R.C.P.,

Physician to the Out-Patients at Westminster Hospital, to the West End
Hospital for Nervous Diseases, and to the Royal National Orthopædic
Hospital.

IN view of the modern localisation of the cortical motor centres entirely in front of the Rolandic fissure, the chief interest of the following case consists in the symptoms associated with a focal lesion of the left post-central gyrus.

The patient was a butcher, aged 51, whose previous history was uneventful. He never had a serious illness or accident, nor was there any history of venereal disease. About 1892 or 1893 he began to suffer from occasional headaches and from constant buzzing noises in the right ear. In 1901 he had paroxysms of intense pain in the right hand, occurring without apparent cause, spreading up to the elbow and lasting about half an hour at a time. These attacks ceased after two or three weeks. In 1904 the headaches became more severe, always unilateral, but not constantly on the same side, starting in the temporo-occipital region of one or other side, and extending up to the vertex. The attacks of headache occurred two or three times a day, lasting for ten or twenty minutes at a time. In the intervals between the headaches he had constant tenderness

over the corresponding side of the head and neck. In November 1905 he had an attack of violent causeless vomiting, lasting for two hours, and another in March 1906. There was no diplopia or affection of vision, nor at this period was there motor weakness of any limb.

He first came under my observation in *April* 1906. At that time all he complained of was diffuse right-sided headache. The optic discs were normal, hearing was slightly less acute in the right ear than the left; the cranial nerves were otherwise normal. The sensory, motor, and reflex functions were all normal on examination. The heart, lungs, abdominal organs and urine were free from signs of disease.

Victoria Saloon
(dictated)

Hospital Sunday
Hospital Sunday (copied)

FIG. 1.—Written with right hand, September 7, 1907.

In *August* 1907 he began to have a difficulty in finding his words. A fortnight later a subjective tingling sensation appeared occasionally in the index and middle finger of the right hand, lasting for five or ten minutes at a time. The headache now became localised to the left parietal region, and his gait became unsteady and staggering. Memory became impaired and speech slow and hesitating, with some indistinctness of articulation. There was no vomiting, no diplopia, and no attacks of unconsciousness or convulsions.

When examined again on *September 7*, 1907, his speech was still slower and more hesitating. In writing he made frequent mistakes of spelling, missing out letters (see Fig. 1). He could understand and execute both spoken and written commands. There was no difficulty in naming objects shown to him. The

optic discs and other cranial nerves were normal save for the slight deafness on the right side previously noted. There was now, however, slight weakness of the right lower face on voluntary movement. There was no anæsthesia or atropognosis. The upper and lower limbs were normal in all movements. The knee-jerks and ankle-jerks were normal and equal; the plantar reflexes could not be elicited. The pulse was somewhat feeble, 100 per minute and of low tension.

By *October 3* the headache in the left parietal region had become much worse. There was no vomiting. The patient was emotional and more deeply aphasic. He had difficulty in finding his words, and spoke with much hesitation. He wrote to dictation fairly well, and could read aloud. The weakness of the right lower face was as before. The optic discs were normal. He was now found to be clumsy with the right upper limb, and seemed disinclined to use it. There was astereognosis of the right hand, as tested with objects such as a watch or a safety-pin. The right grasp was slightly less energetic than the left, otherwise the movements of the right upper limb were normal. The lower limbs were equally powerful. The supinator-jerks and knee-jerks were normal and equal.

On *October 16* the left-sided headache was worse than ever. Speech was still more impaired. The patient had difficulty in finding his words, and sometimes used meaningless syllables. He also seemed mentally dull. The optic discs were normal. There was no hemianopia or restriction of the visual fields.

Motor Functions.—The weakness of the right lower face was as before. There was now total flaccid paralysis of the right hand, and marked, though not total, paralysis of the right elbow and shoulder. In walking, the right leg was now dragged slightly.

Sensory Functions.—Astereognosis of the right hand was still complete. There was no cutaneous anæsthesia or atropognosis. Unfortunately on this occasion joint-sense was not tested. The supinator-jerk and knee-jerk were increased on the right side; there was no ankle clonus. The plantar reflexes could not be elicited. Energetic treatment by mercury and iodide of potassium had been carried out during many weeks.

Under these circumstances the intensity of the headache and the steadily progressive motor aphasia right hemiplegia, in spite of the absence of optic neuritis or vomiting, suggested a

progressive intra-cranial lesion. The patient was therefore sent to Mr Ballance with a view to an exploratory operation over the left hemisphere. The motor aphasia suggested a lesion of the inferior frontal gyrus, whilst the history of sensory fits referred to the right hand, together with the absence of motor convulsions, seemed to point to the post-central gyrus as probably implicated also.

Accordingly, on *October 22*, Mr Ballance performed an osteo-plastic resection of the skull in the left fronto-parietal region. A week later the bony flap was turned down and the dura was opened; it was not adherent. There was no abnormal intra-cranial pressure. The post-central gyrus was seen to be

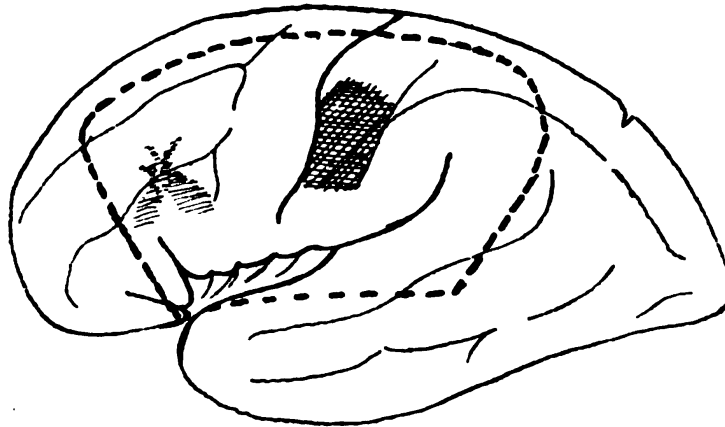


FIG. 2.—Showing position of diseased areas in cortex as seen at operation. The interrupted line indicates the osteo-plastic flap.

markedly diseased in its middle third and adjacent part of the lower third (see Fig. 2), the diseased patch being of a sodden appearance, yellow in colour, destroying the cortex superficially. This yellow area was sharply limited in front by the Rolandic fissure with the vessel coursing therein; posteriorly it was bounded by the intra-parietal sulcus. This area of diseased cortex was removed. In front of the pre-central convolution, which was healthy in appearance, there was an irregular somewhat stellate area of opacity in the pia-arachnoid covering the posterior ends of the second and third frontal gyri, apparently a similar but less active stage of the same process noted in the post-central gyrus. The postero-parietal lobule and the remainder of the post-central gyrus healthy in appearance.

The wound healed uneventfully. On *November 5*, a week

after the operation, speech was still hesitating and the patient had difficulty in finding his words. He could not speak in sentences but only in isolated words, occasionally misplacing syllables. He could read sentences aloud but did not appear to understand them perfectly. He could repeat words and sentences dictated to him and understood vocal commands. With the left hand he could not write spontaneously nor to dictation, but could copy moderately well.

Motor Functions.—There was marked weakness of the right lower face, both at rest and on movement. The external ocular movements were normal and the tongue was protruded straight. There was total paralysis of the right fingers and thumb, flexion of the right wrist was feeble, extension impossible; supination could be performed, but not pronation. The right elbow and shoulder could be feebly moved. There was a moderate degree of atrophy in the interosseal muscles of the right hand. The right upper limb was now slightly rigid at all joints. In walking the right foot was slightly scraped on the ground, but all ordinary movements of the lower limbs were equally performed on both sides in the recumbent posture.

Sensory Functions.—There was no anæsthesia to lightest touches with cotton-wool, or to pin-pricks, on either side of the face, trunk, or limbs of either side. In the right hand there was some atopognosis with cotton-wool touches, the patient referring the touches wrongly in a proximal direction; with pin-pricks there was no atopognosis. Joint-sense was normal at the right shoulder and elbow, lost at the right wrist and digital joints. There was still astereognosis of the right hand, when tested with objects such as a pin, a coin, or a chain. The right supinator-jerk was much brisker than the left; in the lower limbs the knee- and ankle-jerks were slightly increased on the right side; there was no ankle clonus, and the plantar reflexes were both flexor in type.

On *November 22* he was examined again, prior to leaving for his home in the country. Speech had begun to improve. He understood everything that was said to him, but spoke in hesitating fashion, mostly in single words, in "telegraphic" style. He could read aloud simple sentences such as "Shut your eyes," but did not always attempt to carry them out, though sometimes he executed written commands such as "Put out your tongue,"

"Smile," "Give me your left hand." He could repeat sentences dictated to him fairly well, but occasionally misplaced syllables. He could sing songs; for example, "Auld Lang Syne" or "God Save the King," the airs being correctly rendered, but some of the words were occasionally repeated twice. He read the daily papers and understood the news. When asked what he had been reading, he replied, "Cricket match, Australia, draw" (all of which statements were correct).

Motor Functions.—Weakness of the right lower face was still present on voluntary movement; there was no difference on emotional movements. He could feebly move the right upper limb at all joints; the right grasp was very feeble. The interosseal wasting was still well marked. The lower limbs seemed equally powerful in the recumbent posture, but in walking the right foot was slightly scraped.

Sensory Functions.—He could feel the lightest cotton-wool touches and pin-pricks equally acutely on both sides of the face, trunk and limbs, except on the right hand, where touches seemed slightly less distinct. Joint-sense was normal at the right shoulder, elbow, and wrist, lost at the digital joints. Astereognosis of the right hand was as before, the patient failing to recognise a watch, a pin, a shilling, a key or a chain, by touch, all of them being promptly recognised by the left hand and correctly named.

The right supinator-jerk was markedly increased, the right knee and ankle-jerks slightly brisker than on the left side; the plantar reflexes remained flexor in type. The abdominal reflexes were absent.

The patient was examined again on *May* 6, 1908, over five months after the operation, when his condition was as follows:—

He understood everything said to him; he also read intelligently letters and newspapers. When given written requests, he read them aloud before executing them. Spontaneous speech was still hesitating and with occasional mistakes of pronunciation, *e.g.* "sesterday" instead of "yesterday." He repeated correctly sentences dictated to him. He wrote fairly well with the left hand, both to dictation and on copying, and was able to add up accounts (see Fig. 3).

Motor Functions.—There was still slight weakness of the right lower face. He could execute all movements of the right

upper limb, not quite as powerfully as with the left. Part of the impaired movement of the limb seemed due to adhesions in the right shoulder-joint. The distal joints were less arthritic, and

Hospital
HOSPITAL

copied.

thank

asked to write his Christian name

Buster

dictated.

Brighton

dictated.

Logan

Asked to write down when he came.
He replied "yesterday," but could not write it.

1	2	12	-	
	1	13	6	
<hr/>				
4	5	4		

Addition sum.

FIG. 8.—Written with left hand, May 6, 1908.

the interosseal atrophy which followed the operation had now disappeared. The lower limbs were both powerful and the gait practically normal.

Sensory Functions.—Cotton-wool touches and pin-pricks were felt equally on both sides on the face, trunk, and limbs. There

were occasional mistakes in topognosis in the right fingers. Heat and cold were equally felt on both sides. Joint-sense was impaired in all the digits, less impaired in the thumb than in the other fingers; it was normal at the wrist, elbow, and shoulder. Astereognosis was still complete in the right hand, when tested with a bottle, a watch, a chain, a pencil, and a penny. He recognised all these objects promptly with the left hand and named them.

The deep reflexes in the right upper limb were markedly increased; in the right lower limb they were slightly brisker than on the left side. There was no ankle clonus and the plantar reflexes were flexor in type. The abdominal reflexes were still absent.

On May 8 the patient was shown at a meeting of the Clinical Section of the Royal Society of Medicine.

The termination of the case was independent of his cerebral condition. About the middle of May the patient developed acute right-sided abdominal pain, together with vomiting and collapse. Medicinal means having failed to relieve him, laparotomy was performed by Mr Ballance on the 15th May, the gall-bladder and right kidney being explored with negative results. The patient died on May 17.

The autopsy was performed by Dr C. R. Box, to whom I am greatly indebted for the following note of the conditions found post-mortem.

The surface of the left cerebral hemisphere had been exposed and explored six and a half months before death. The upper part of the large osteoplastic flap had sunk inwards for half an inch. The dura was closely adherent under the flap, and the brain was extricated with difficulty, undergoing some laceration in the process.

There was considerable shrinkage of the convolutions of the convex aspect of the left frontal lobe and of part of the parietal lobe, as indicated in the diagram. When the pia-arachnoid was stripped off the shrunken convolutions, they were found to present a bright yellow discoloration. This pigmentation was extensive and penetrated in some places for quite quarter of an inch; it was in the convolutions, not in the membranes, and appeared to be the residue of an old extensive superficial red softening which had occurred some time previous to the operation. Immediately subjacent to the lower and upper parts of the wasted area there was also some softening of the white matter

but it did not extend to any great depth. Owing to the shrinkage of the cortex, the Sylvian fissure was opened up and the insula exposed (see Fig. 4).

The arteries of the base were quite healthy. The trunk of the left middle cerebral was slit open, up to the area of atrophy, but no obstruction was discovered, nor any trace of past disease. The artery could not be traced over the shrunken area owing to the thickening and matting of the membranes. The wasted area

Rol.

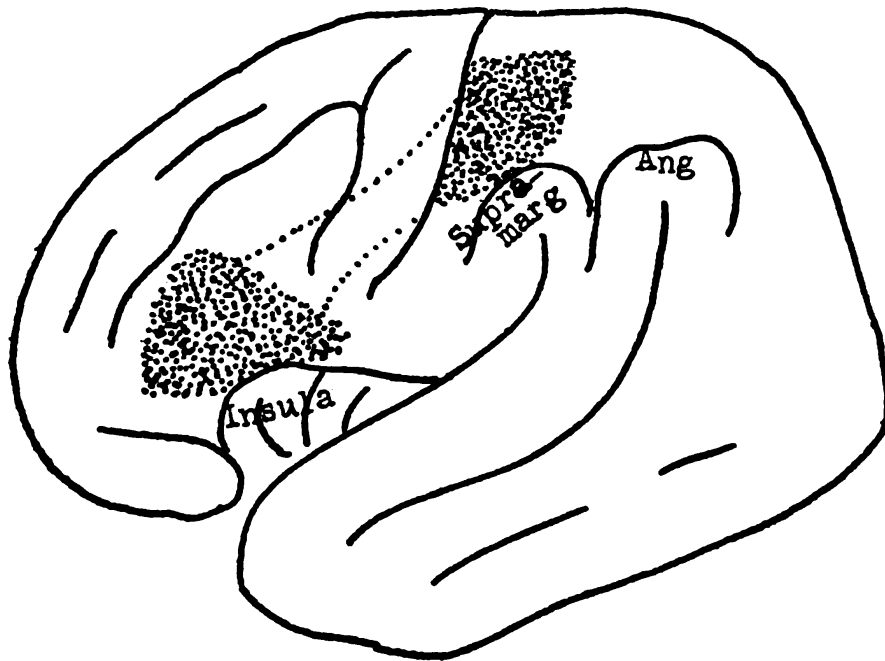


FIG. 4.—Diagram of condition post-mortem, after removal of the membranes. The two areas of superficial softening are indicated by dotted shading.

was entirely in the domain of the middle cerebral artery, but did not involve the whole of its distribution.

The basal ganglia of both sides appeared healthy. Coronal sections, after hardening, showed that there was no implication of the white matter of internal or external capsule by the softening process. There was no evidence of disease of the right hemisphere.

The mouth, fauces, larynx, pharynx, bronchi and oesophagus were healthy. There was an extensive and very acute pleurisy with turbid exudation on the right side, where there were also some old basal adhesions. The lower lobe and part of the upper lobe showed extensive collapse, and, in addition, near the root of the lung, were some patches of recent broncho-pneumonia. The left pleura was healthy, and the left lung highly oedematous from apex to base. The pericardium contained a slight excess of

serum. The myocardium was soft, friable, and light brown in colour; there were no valve lesions; the right auricle was distended with recent clot.

There was an extensive sub-peritoneal blood-extravasation encasing the right kidney. This kidney had been explored and sutured; its pelvis was distended with red blood-clot. The supra-renal capsules were moderately adherent, and there was a slight but definite degree of cortical atrophy in each. The small renal arteries were thickened. The spleen was greatly enlarged and very flabby, its parenchyma being semi-diffuent. No lesions were discovered in the intestines.

As regards the aphasic phenomena in this case, it should be noted that the third left frontal gyrus was extensively diseased. The patient was slightly dull mentally, and his mistakes in spoken and written speech consisted mainly in impairment of words spoken or written by himself. Cortical word-memories being impaired, he had difficulty in understanding complicated sentences, whether spoken or written; he also had a degree of motor agraphia proportional to his vocal speech-defects. He was neither word-deaf nor word-blind.

The absence of motor convulsions was doubtless due to the absence of disease of the cortical motor cells of the pre-central gyrus. The marked monoplegia of the right upper limb which preceded the operation was at first sight difficult of explanation, in view of the superficial integrity of the pre-central gyrus at the operation. At that time I suggested that there might be a focus of sub-cortical disease implicating the brachial pyramidal fibres in their downward course. Autopsy proved this surmise to be correct, inasmuch as there was a narrow band of partial softening crossing the pre-central gyrus (see Fig. 4).

It is interesting to note that the paralysis of the upper limb was flaccid in type before operation, but subsequently became spastic, with increase of deep reflexes. The transient atrophy of the intrinsic hand muscles, which followed the operation, is also interesting and at present unexplained.

Perhaps the most interesting features of the case are its sensory phenomena. These may be divided into—(1) an irritative, and (2) a paralytic group.

(1) The irritative phenomena consisted in paroxysms of pain in the right hand, constituting the earliest symptom of the

disease some fifteen years ago. Two months before operation paroxysms of tingling reappeared in the index and middle finger of the right hand and coincided with the rapid advance which then supervened in the other symptoms. We are, I believe, justified in regarding these symptoms as a variety of sensory fit, due to the irritative lesion of the post-central gyrus. An almost identical case was recorded by Mills and Weisenburg¹ in 1906, where autopsy showed an area of syphilitic disease in the post-central gyrus, extending slightly across the Rolandic fissure to the precentral gyrus and also partly into the inferior parietal gyrus.

(2) The destructive phenomena comprised atognosis and loss of joint-sense in the right hand, with astereognosis. These symptoms appear to be the result of the lesion of the post-central gyrus. The sensory area for the upper limb in the post-central gyrus seems to correspond in position with the motor centre in the pre-central gyrus, the two centres facing each other, as it were, on opposite sides of the Rolandic trench.

Loss of joint-sense and atognosis are fairly well recognised symptoms of cortical lesions in the central convolutions. As to the question of astereognosis, some writers, notably Mills and Weisenburg, have endeavoured to limit the astereognostic function to one special cortical area, separate from the other cortical centres for cutaneous, joint, and muscle-sense. Mills and Weisenburg go so far as to suggest the postero-parietal lobule as the so-called "stereognostic centre," quoting in support of their view the case of lesion in that region, to which reference has already been made. But we should note that the faculty of stereognosis, or the recognition of the shape of solid objects, is not a primitive sensation, but a complex psychical process, which is arrived at by the combination and comparison of various sensory impressions:—cutaneous, muscular, and articular, from the hand. Moreover, stereognosis implies the recalling of visual and other memories and a comparison of these memories with the actual sensations felt at the moment in the hand. If any of these primary sensations be absent, a stereognostic judgment may be impossible, even though the cortex be intact. There are many morbid states in which we meet with the

¹ *Journal of Nervous and Mental Diseases*, 1906, p. 617.

clinical syndrome of astereognosis. No case of astereognosis has been observed without impairment of some of the primary sensations. Thus, for example, it is a common phenomenon in cervical tabes, where the patient complains that he cannot recognise objects, such as coins or keys, in his pockets. Again, in some lesions of the optic thalamus, astereognosis in the contra-lateral hand is well marked. And in cortical lesions of the sensory areas, as in the present case, there may be complete astereognosis. In this case the postero-parietal lobule was intact, the lesion not extending behind the post-central gyrus. There is, therefore, no need to postulate a special stereognostic centre, whether in the parietal lobe or elsewhere.

A CASE OF SPASMODIC SYRINGOMYELIA (?).

By ALEXANDER BRUCE, M.D., F.R.C.P.E.,
Physician to the Royal Infirmary, Edinburgh.

C. G., aged 50, unmarried, employed as a time-keeper on the Forth Bridge, was sent to me by Mr Cathcart on account of a peculiar flexion of the ring and middle fingers of the left hand and of a heaviness and rigidity of the left arm.

When I first saw him in my waiting-room, my attention was arrested by his remarkable attitude. A powerfully built, healthy-looking man, he sat with his left arm flexed at the elbow and supported by his right hand, his left hand presenting an appearance recalling that of the "finger-post" of the sign-painter, the two middle fingers firmly flexed in the palm, the fore-finger and thumb pointed almost straight forward, and the little finger very slightly flexed. The position is indicated in Fig. 1, from a photograph which was taken some time subsequently.

The attitude at once recalled to me that shown in the illustrations in Guillain's thesis on the spasmodic form of syringomyelia (1), first noted by Pierre Marie. In view of the rarity of this condition, and on the possibility that this may be an early example of it, the following notes of the case may probably prove of interest.

Previous Occupations.—The patient had been employed as a

farmer from the age of 16 to 22, as a law clerk from 22 to 27, as a labourer from 27 to 43, and since that time he has acted as a watchman or time-keeper on the Forth Bridge, in which capacity he has undoubtedly been exposed to much severe weather.

His previous health has been uniformly excellent, apart from a compound fracture of both bones of the right leg caused by the fall on it of a sugar barrel twenty-four years ago. Examination of the cardiac, respiratory and genito-urinary systems showed them to be quite normal. His family history reveals nothing that can have any possible bearing on the case.

His present illness appears to have commenced about the beginning of 1907. During the month of February he observed that when at work objects would fall from his left hand in an unaccountable way. He stated that his left hand became numb, but on closer questioning it was found that there was at first no real numbness, but rather a curious incapacity to estimate and regulate the amount of pressure necessary to hold an object in the hand. He said that even when he felt as if an object were about to fall from his hand and endeavoured to grasp it tightly, it might drop notwithstanding this attempt. He could not hold anything in the unaided left hand for any length of time. Apart from this apparent loss of muscular sense, or loss of the power to regulate the force of the contraction of the muscles of the hand, there was at first no stiffness or malposition of the fingers, and no real weakness in the hand. After this condition had lasted for between two and three weeks, a slight numbness was noted along the ulnar side of the forearm and hand, and shortly afterwards he wakened one morning to find that the middle and ring fingers of the left hand were firmly flexed into the palm, and that the position of the fingers, thumb and hand was much as is shown in Fig. 2, and as will now be described. Shortly after this the rigidity of the arm and forearm appear to have developed, but its onset did not attract his immediate attention.

Position.—The patient always supported the left forearm with his right hand, because, as he said, "the left arm felt heavy and the shoulder dragged."

The arm was apposed to the side of the thorax, the elbow flexed at nearly a right angle, the shoulder slightly raised and drawn forward, and the hand held either pointing forwards or

across the trunk (Fig. 1). The wrist was slightly over-extended ; the thumb and fingers, as already said, presenting almost the position of a finger-post, the thumb and forefinger being extended in a direction almost parallel to each other, the ring and middle finger firmly flexed, and the little finger partially flexed.

The ring and middle fingers were firmly flexed at the metacarpo-phalangeal and proximal inter-phalangeal joints, and extended at the distal inter-phalangeal joints. The pulps of the finger-tips were firmly pressed into the palm. The little finger was usually kept slightly flexed at the metacarpo-phalangeal joint, and almost fully extended at the two inter-phalangeal joints.

The index finger was slightly flexed at the metacarpo-phalangeal joint, and was completely extended at the two inter-phalangeal joints. It was slightly adducted towards the ulnar side.

The thumb was almost parallel and close to the index finger. Its metacarpo-phalangeal joint was straight ; the inter-phalangeal joint was slightly over-extended. The thenar eminence was of normal size ; it was slightly drawn inwards (in opposition), and the tip of the second finger lay on its base. The interosseous spaces and the hypothenar eminence were normal.

MOVEMENTS.

(a) *Voluntary* :—

The little finger was capable of slight voluntary movements of extension, flexion, and of abduction and adduction at the metacarpo-phalangeal joints. On one occasion patient succeeded in feebly flexing the finger and maintaining it in a position of flexion for some time.

The ring and middle fingers.—With great difficulty the tips of the fingers could be raised half an inch from the palm.

The index finger could be slightly extended and flexed at the metacarpo-phalangeal joints, but not at the inter-phalangeal joints. It could not be adducted or abducted.

The thumb could be slightly flexed and extended at the metacarpo-phalangeal and inter-phalangeal joints. When the second and third fingers were passively raised from the palm, patient could slightly abduct the thumb.

(b) *Passive*:—

The little finger was capable of a wide range of passive movement, the only limitation being due to the flexed position of the second and third fingers. It could be flexed and its tip placed below that of the ring finger. It could be adducted and abducted, and there was no feeling of traction resistance to any movement. When the finger had been flexed on the palm, it immediately sprang back to its usual position when the pressure was removed.

The ring and middle fingers.—The attempt to extend these fingers was met with a steady and increasing resistance, due either to involuntary spasm or to shortening of the muscles. As the limit of possible movement (about one inch of the tips from the palm) was reached, pain was complained of in the flexor muscles of the fore-arm and in the palm of the hand, but the joints were in no way painful. The flexor tendons in the palm stood out prominently as in a case of shortened muscle. As soon as the traction on the fingers was removed they recoiled to their previous position. There was neither general nor local contraction of the palmar fascia.

The index finger.—Passive movement was possible over a considerable range, and the degree of resistance was very much less than in the case of the second and third fingers.

The thumb could be flexed and extended at all its joints, and abducted, adducted, and opposed at the carpo-metacarpal joint. There was some resistance to its movements, but much less than in the case of the second and third fingers.

Wrist.—The wrist was generally maintained in a position of slight hyperextension. It could be slightly extended and flexed, but with great difficulty. The flexion could not be carried further than to put the back of the hand and forearm on a straight line. Relaxation was followed by an immediate recoil to a position of rest in extension. The extension could be slightly increased. The difficulty of movement was obviously due not to any lesion of the joint, but to muscular resistance. One had the feeling as if the muscles were too short to permit of the proper range of movement. While moving the wrist the muscles appeared to be put into a condition of spasm. Very slight ulnar and radial deviation were possible. Voluntary movement was greatly limited. Passive movement produced

pain in the muscles of the forearm when the limit of the possible movement was approached.

Elbow.—The elbow was kept flexed at an angle of 135 degrees. It could be passively extended to about 145 degrees, and could be flexed to 70 degrees. Voluntary movement had a range of about 45 degrees, flexion being possible to 75 and extension to 120. Slight pronation and supination were possible.

Shoulder.—The upper arm was kept closely apposed to the side of the chest. The shoulder was slightly raised and drawn forward. The arm could be abducted passively to about 45 degrees. When this angle was reached the triceps muscle passed into spasm, and further abduction became impossible. Anterior and posterior movements were greatly restricted, and voluntary movement was very slight.

There was a slight curvature of the spine, the convexity being to the left in the cervical, and to the right in the lower thoracic region.

It was repeatedly noted that the degree of muscular rigidity, whether estimated by direct palpation of the muscles or by the resistance to passive and voluntary movement, varied within considerable limits from time to time. At one time the muscles might yield before the hand fairly easily; at another they could not be compressed at all.

It was generally noted that when the muscles were in a condition of comparative relaxation, any attempt at passive movement of them almost at once brought about the state of spasmodic rigidity. A similar result followed when they were massaged.

He slept as a general rule lying upon his left side, the weight of the body resting to a certain extent upon the left elbow. The forearm stood up so as to form about a right angle with the upper arm, and was almost invariably left outside the bed-clothes. It was generally supported by the right hand. This was noted during the whole of his residence in hospital. His attitude might occasionally be varied, the patient lying on his back and right side, but this did not often happen. The attitude of the fingers remained the same as that noted when the patient was awake, and any attempts—made so as not to waken him—to straighten the fore and middle fingers met with the same resistance as was present during the day.

The right arm, forearm, and shoulder appeared larger than the left. The measurements were :—

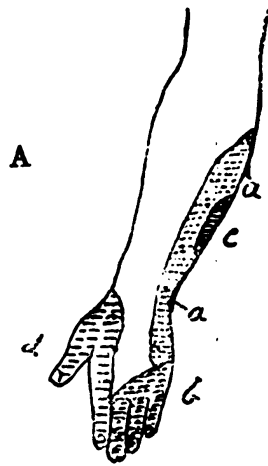
Right Arm.		Left Arm.	
Wrist . . .	7½ inches.	Wrist . . .	7⅔ inches.
Middle forearm .	9⅔ „	Middle forearm .	10¼ „
Below elbow .	11 „	Below elbow .	11¼ „
Above . . .	10¼ „	Above . . .	10¾ „
Middle upper arm	11¾ „	Middle upper arm	12¾ „
Just below axilla .	13¼ „	Below axilla .	13¼ „

Reflexes.—The deep reflexes in the left upper extremity were all greatly affected, the supinator and biceps being both absent, and the triceps-jerk slight. This abnormality appeared to be due to the rigidity of the opposing muscles. The deep and superficial reflexes were normal in the right upper extremity and in both lower extremities.

Electrical Reactions.—These were examined by Dr Harry Rainy, and were found to be somewhat peculiar. There was no true reaction of degeneration, but both the faradic and galvanic reactions were reduced. With both forms of stimulation much stronger currents were required to produce a reaction than over the corresponding nerves and muscles on the right side, and the resulting contraction was less in extent than on the opposite side. On galvanic stimulation of the muscle there was no alteration of the polar reaction. One's impression on making the examination was that the electric current was diffused by the infiltrated tissue on the left side, and that the muscles, being already in a high degree of spasm, and being powerfully resisted by the opponent muscles, were not in a condition to contract fully. Currents of such strength as to be decidedly painful did not alter the character of the contractions.

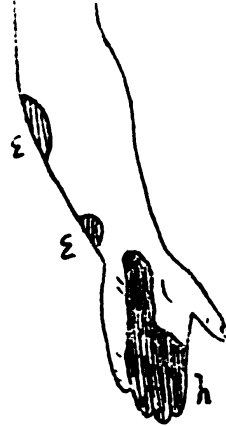
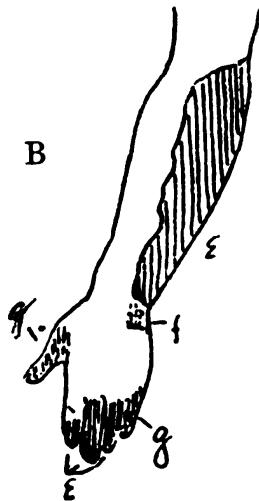
All forms of sensibility were normal in the right arm, the head and trunk, the lower extremities, and in the left upper arm.

Sensibility.—The examination of the various forms of sensibility of the left forearm was a matter of considerable difficulty, as patient's answers varied a good deal from day to day, and indeed during a single examination. Repeated and careful examinations by Dr Kelman Macdonald elicited the fact that there were more or less constant departures from the normal, which were much as represented in the annexed figures.



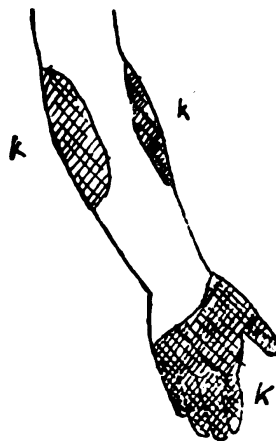
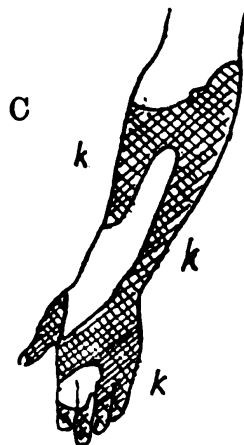
A. Sensibility to Touch.

- (a) Areas of diminished perception.
- (b) Areas of erroneous localisation.
- (c) Area of complete anaesthesia.
- (d) Area where touch perceived but not localised.



B. Sensibility to Pain.

- (e) Areas of complete analgesia.
- (f) Area of diminished perception.
- (g) Areas of erroneous localisation.
- (h) Area of hyperalgesia.



C. Sensibility to Heat and Cold.

- (k) Areas of complete absence of perception.

After the date at which the chart was taken, of which the figs. are a reduced copy, no further reference to the sensibilities was made in the patient's presence, and no examination was undertaken until a period of six weeks had elapsed. Then on a fresh examination nearly the same condition was ascertained to be present. It will be seen from a comparison of the figs. that the disturbances were in each instance mainly on the ulnar side of the forearms and on the hands.

It will be seen also that there is a preponderance of loss of sensibility to pain and to temperature as compared with touch—an imperfect dissociation symptom.

It is possible that the variation in the results of the examinations may have been due to the overlapping of the various areas of the distribution of the adjacent nerves, which may have allowed a strong stimulus to be perceived by the nerves from the adjoining area although they may not have been capable of perceiving a slight impression.

On palpation it was felt that the right shoulder, pectoral and scapular regions, as well as the upper arm and forearm, had a peculiar firm, brawny feeling, somewhat recalling that of rigor mortis, or of an early stage of sclerodermia with fibro-myositis. On endeavouring to pinch up the skin over the subjacent tissues in the above-mentioned regions, one could not catch hold of it so easily as on the corresponding parts on the opposite side of the body. Either the fingers slipped over the skin, or they had to grasp a larger mass of tissue in order to raise the skin. This arose evidently from infiltration of the cutis vera and subcutaneous tissues. On pressing deeply, one felt also that the muscles were unduly firm.

The boundaries of the area of infiltrated skin were not sharply marked off from those of normal skin, but they corresponded fairly well to a line drawn horizontally outwards from the apex of the axilla to the spine, and from this point upwards to the vertebra prominens. In front the area corresponded, as far as could be estimated, to the pectoral region. The whole of the upper arm showed much the same condition; but the infiltration of the skin and subcutaneous tissues was somewhat less dense.

The skin of the fingers, especially of the forefinger, showed a markedly glossy and slightly livid appearance. So also did the

ulnar border of the little finger, on which there were one or two small recent cicatrices. The nails appeared normal.

The left pupil was somewhat larger than the right, and it dilated in a dim light to a considerably greater extent than did the right one. When contracted by a strong light, however, they were almost equal.

The mental condition of the patient was that of an intelligent working-man. He impressed one as a man of sound common-sense and judgment, and did not betray any neurotic tendency except some depression such as was natural from the dread that his condition was incurable.

Progress.—The patient remained in Hospital from 3rd December 1907 to 29th February 1908. The affected arm and shoulder were treated with massage and electricity, ionisation with chloride of sodium from the negative pole, and with static electricity (by Morton's wave-currents). The result was a considerable diminution of the infiltration of the skin and subcutaneous tissues of the arm, shoulder and back. A certain diminution of the rigidity of the muscles was also noted, both voluntary and passive movements at the various joints becoming somewhat more free than on admission. The arm could be voluntarily abducted from the side to 45 degrees; the elbow could both passively and voluntarily be almost straightened. Movements at the wrist were also improved, but to a much lesser degree. The fingers could be passively separated from the palm for a distance of about two inches, but they recoiled to their initial position almost immediately after the traction was removed.

The left biceps muscle appeared to increase steadily in size, and all those who saw it were much struck by the contrast between it and its fellow. It stood out in relief, as is shown in Figs. 3 and 4, and on palpation it was evidently larger and much firmer than the right one. It was also noted that massaging the muscles of the left arm and forearm rapidly made them harden up, and on several occasions it appeared that the muscles of the right upper arm tended to become too readily firm when they were massaged, as if there were a slight degree of spasm. There has, however, been as yet no appearance or suggestion of the occurrence of any further change involving the right arm.

A CASE OF SPASMODIC SYRINGOMYELIA (?) 399

The sensory condition has remained practically unaltered.

The gait has remained unaltered. The reflexes in the lower limbs are not exaggerated or in any way modified from the normal. The head droops somewhat forwards. The back is slightly rounded, and there is the same degree of scoliosis as was noted at first. The left shoulder is always slightly raised, and the free edge of the trapezius somewhat unduly prominent.

The *diagnosis* is a matter of great difficulty. The most probable one is that of a very early case of the spasmodic form of syringomyelia, first described by Pierre Marie in 1900. It was suggested that the condition might be a purely functional one, and every reasonable care was taken to exclude this possibility. The following facts seem to militate against this view: (1) the steady persistence of the condition of the left hand and arm during sleep; (2) the failure at any time to find a relaxation of the spasm when the patient thought he was not under observation; (3) the mental character of the patient, which was one obviously of sound judgment and common sense, without the slightest suggestion of any neurotic element; (4) the condition of the skin and sub-cutaneous tissues of the left arm and shoulder, as well as the cyanosis of the forearm and the glossy skin of the fingers; (5) the greater circumference of the left arm as compared with the right, and the gradual increase under observation of the firmness and size of the left biceps muscle; and (6) the character of the electrical reactions.

The spasmodic form of syringomyelia, which was first described by Pierre Marie at a meeting of the Faculty of Medicine in Paris in 1900, and which has been further studied at the Bicêtre Hospital by his pupil, G. Guillain, presents in its fully-developed condition the following symptoms:—

(1) The arms are firmly apposed to the anterior part of the side of the thorax, the hands almost meeting in front of the pubis, the shoulders raised and inclined forwards and downwards (*thorax en bateau*), and the back rounded (somewhat as in paralysis agitans).

(2) The hands show a marked spastic flexion of the three last fingers into the palm, while the thumb and forefinger remain extended and slightly flexed towards each other like a pincers (*main en pince*) and are capable of being used as such.

(3) The upper limbs are in such firm spasm that they can scarcely be moved. The lower limbs are markedly spastic.

(4) The deep reflexes are exaggerated, and the plantar reflex gives an extensor response.

(5) The disease may develop one or many years after an injury, or may come on without cause.

(6) The evolution is slow, and the disease may last for more than twenty years, proving fatal from some complication, such as bladder trouble, ulcerative cystitis readily developing and frequently leading to septic infection. Death may arise also from broncho-pneumonia or general nervous exhaustion.

Guillain has collected five cases in his thesis, and has described two additional cases in conjunction with Alquier (2) and Raymond (3). Raymond and Français (4) report an eighth case, and we have the record of a doubtful case by Verger (5).

A comparison of my case with those described by Guillain shows that the condition of the left hand and upper extremity corresponds almost exactly with that illustrated by his figures. The position of the hand is almost identical, with the exception of the fact that the little finger is not flexed into the palm, as in all his cases. The position of the forefinger and thumb, with the ability to use these to a certain extent, is quite similar. There is the same hyper-extension of the wrist, and the same resistance to both passive and active movement—a resistance obviously due to muscular spasm. (It is somewhat to be regretted that Guillain has not given a more precise description of the condition of the muscles themselves.) There is the same tendency for the fingers to recoil like a spring after they have been forcibly extended, and the same tendency to irregular spasmodic action of the hand at an early stage, as noted in his case 3.

The indefinite, varying dissociation symptoms are also somewhat similar to those described in one of his cases.

On the other hand, it is to be noted that there is no interference with the gait, with the tonicity of the muscles of the lower extremities, nor with their deep and superficial reflexes, and that there is no bladder trouble. These facts would, of course, be opposed as much to the diagnosis of pachymeningitis with spasm as to that of spasmodic syringomyelia.

I have searched in vain the accessible literature of sclero-



FIG. 1.



FIG. 2.



FIG. 3.



FIG. 4.

dermia and myosclerosis for evidence that they may be associated with muscular spasm, such as presented by my patient. It must apparently be one of an early stage of spasmodic syringomyelia in which one limb is as yet alone affected. If not, it must be some independent, and as yet apparently undescribed condition.

I have in conclusion to thank Dr Harry Rainy, Dr Hugh More, and Dr Kelman Macdonald for valuable assistance in the study of this case.

LITERATURE.

1. Georges Guillain. "La Forme Spasmodique de la Syringomyélie." Steinheil, Paris, 1902.
2. Alquier et Guillain. "Étude anatomo-clinique d'un cas de Syringomyélie Spasmodique." *Rev. Neurol.*, 1906, p. 489.
3. F. Raymond et Guillain. "Un Cas de Syringobulbie. Syndrome d'Avellis au cours d'une Syringomyélie Spasmodique." *Rev. Neurol.*, 1906, p. 41.
4. Raymond et Français. "Syringomyélie Spasmodique avec Attitude Particulière des Membres Supérieurs." *Rev. Neurol.*, 1906, p. 350.
5. Henry Verger. "Sur un cas de Syringomyélie Spasmodique Douleur-euse à évolution rapide." *L'Encéphale*, 1907, p. 21.

DESCRIPTION OF PLATES.

FIG. 1. Usual attitude of patient. Left arm firmly apposed to side of thorax. Forearm flexed at a right angle to arm and supported by left hand. Slight hyperextension of wrist. Flexion of middle and ring fingers. Slight flexion of little finger. Forefinger extended at two interphalangeal joints and flexed at metacarpo-phalangeal joint. Left shoulder somewhat higher than right. Greater volume of left shoulder and arm.

FIG. 2. Attitude of left hand.

FIGS. 3 and 4. Side views of left and right upper limbs to show the prominence of the left biceps muscle.

Abstracts

PHYSIOLOGY.

THE "FLY-CATCHING REFLEX" IN THE FROG. J. A. GUNN,
(341) *Quart. Jour. Experiment. Physiol.*, Vol. i., No. 2, April 1908.

SCHRADER has shown that in the frog the snapping for food is a reflex from sight stimulation, and that a frog deprived of its cerebrum will catch flies under suitable conditions. He has also shown that if the brain be destroyed down to the fore part of the

medulla oblongata, a somewhat different snap reflex is developed, the frog in this case snapping if his hand or nose be touched. The author points out that similar reflexes occur in a frog poisoned with yohimbine, namely, it snaps if its nose or hand be touched, or if a bright object be brought near its nose.

He considers that these reflexes are produced by yohimbine, either by its paralysing the upper part of the central nervous system and simulating the effects of operation, or by its action on the medulla oblongata facilitating the elicitation of a normally latent reflex. There are evidences in favour of both explanations. However produced, the phenomenon is a striking illustration of the selective action of a toxic agent on the nervous system.

AUTHOR'S ABSTRACT.

ON THE STRUCTURE AND CONDUCTION OF SUPRANUCLEAR

(342) AUDITORY TRACTS. (Ueber Bau und Leitung der supranuklearen Hörleitung.) ROTHMANN, *Beitr. z. Anat., etc., des Ohres.*, Bd. 1, H. 3, p. 232.

THE paper is an abstract of the literature on this subject. The first part deals with the anatomy, and is too detailed to abstract. The second portion is concerned with the physiology. The conclusions reached are that unilateral destruction of the central auditory tracts in no case causes unilateral deafness. The bilateral destruction of the posterior corpora quadrigemina interferes greatly with the hearing, but permits of a portion of the auditory fibres reaching the cortex. Bilateral destruction of the corpora geniculata interna entirely destroys the hearing. In the cortex only bilateral destruction in the region of the temporal convolution will lead to permanent loss of hearing in dogs and monkeys.

W. G. PORTER.

AXON BIFURCATION IN REGENERATED NERVES. W. A.

(343) OSBORNE and BASIL KILVINGTON, *Journ. Physiol.*, Vol. xxxvii., No. 1, p. 1.

WHEN an efferent axon is cut and offers more than one path along which to regenerate, Langley and Anderson showed that it can divide into at least two separate and distinct axons. The authors of the present paper find that the axon bifurcation takes place, not at the region where the regenerating stump is sprouting, but at the point where the multiple path is offered.

If a sensory path be offered to a regenerating motor nerve, as well as its own proper path, bifurcation takes place just as if both

the offered paths had been motor. One branch of the bifurcating axon reaches the proper ending in the muscle, the other branch travels down the sensory path, and can be detected as far as the stimulation test can be applied.

In the adult dog afferent nerve fibres cannot regenerate along motor paths. One case, however, seemed to be an exception to this rule. The radial nerve was cut (in a dog), about an inch of the posterior interosseous nerve was excised, and then the central end of the radial was sutured to its own distal end and to the distal end of the posterior interosseous. There was thus a motor (posterior interosseous) and an afferent (radial) path offered to an afferent nerve (radial). Stimulation was carried out 113 days after the primary operation. The radial was cut $1\frac{1}{2}$ inch above the point of suture, and on being stimulated on the peripheral side of this point marked contraction of the extensors was produced. The radial was then cut distal to the point of stimulation, but still contraction of the extensors was obtained. On dividing the posterior interosseous no contraction was caused by stimulating the radial either peripheral or central to the suture. The radial nerve, though it contained no fibres carrying nerve impulses into muscle cells, could nevertheless, when offered a motor path along which to regenerate, supply this motor path with efficient axons. The explanation offered by the authors is that in this particular instance motor fibres were actually present in the radial, though incapable of making any peripheral muscular connection, the aberrant course being due to a developmental anomaly.

If a deficient sensory path is offered to regenerating sensory fibres, no coalescence of sensory axons can be detected.

SUTHERLAND SIMPSON.

ON RECIPROCAL INNERVATION OF ANTAGONISTIC MUSCLES.

(344) **ELEVENTH NOTE—FURTHER OBSERVATIONS ON SUCCESSIVE INDUCTION.** C. S. SHERRINGTON, *Proc. Roy. Soc.*, Vol. lxxx., p. 53.

In a spinal animal after a sufficient interval has elapsed to allow of recovery from "shock," or in a decerebrate animal (transection through anterior part of hind-brain) within a few hours after the operation, the "flexion-reflex" obtained from the limb by excitation of an afferent nerve or of some appropriate skin-point is found to be diphasic; the movement of active flexion is followed by a movement of active extension. During the active flexion the extensor muscles are relaxed by central inhibition; during the active extension the extensor muscles contract. This is termed "successive induction."

The contraction of the extensor muscles never occurs *during* the continuance of the external stimulus, but only after that has ceased entirely or been greatly reduced in intensity, and only then if the original stimulus is stronger than a certain minimal. The intensity of the contraction after-phase is proportional, within limits, to the strength and duration of the stimulus.

In the first phase of the reflex the extensor muscles abandon the maintenance of a posture, or the execution of a movement in which they were engaged; in the second phase they restore that posture to the limb, or re-establish movement in the abandoned direction. The active movements, therefore, of flexion and extension alternating one with another do not require alternation of two external stimuli, one evoking flexion, the other extension; one and the same stimulus intermittently applied or merely suffering periodic variations of intensity, provided the variations exceed a certain amount, suffices fully for the double phases of the reflex movement. The reflex movement of stepping, therefore, with its two opposite phases of flexion and extension, is excited by one single form of stimulus, that stimulus being the one which directly excites flexion. This suggests an explanation of the fact that flexion is much more extensively represented in the receptive field of the limb than extension. The direct stimulation of any afferent limb nerve excites as its immediate result flexion of the limb itself, not extension. Similarly in the motor cortex, especially for the hind limb, the primary representation of flexion greatly preponderates over that of extension, the reason being that reflex flexion of the limb of itself induces as a sequence extension, so that no local stimulus is required for extension.

SUTHERLAND SIMPSON.

PATHOLOGY.

CONTRIBUTIONS TO BRAIN PATHOLOGY. (*Beiträge zur Pathologie (345) des Gehirns.*) HOCHHAUS, *Deutsche Zeitsch. f. Nervenheilk.*, 1908, Bd. xxxiv., p. 185.

THE first case here recorded was one of multiple gliomata of unusual magnitude. The main tumour was of enormous size, and there were two smaller accessory gliomata. The disease commenced in March 1901 with headaches, giddiness, and impairment of vision. Under treatment by potassium iodide, with mercurial inunction, there was temporary improvement. But after a year the same phenomena reappeared in more marked degree. The patient became blind from optic atrophy, paresis of external ocular muscles

developed, and in August 1902 paralytic phenomena appeared in the left trigeminal area and in the left side of the body. Attacks of unconsciousness, with convulsions, developed, and after various remissions and exacerbations the patient died in August 1903. At the autopsy the right side of the brain was increased in volume, especially in the region of the temporal lobe, from which a gliomatous tumour projected downwards, compressing the right crus cerebri. The infundibulum and optic chiasma were embedded in tumour-tissue. The main tumour measured nearly 20 cm. in length, occupying the whole right hemisphere. Two smaller tumours were also found, one in the right temporal lobe, the other in the right frontal lobe. These smaller tumours were independent of the main mass of the growth, though of the same microscopic structure.

The second case was one of infantile hemiplegia in a child aged 2½ years. After measles the child was suddenly taken ill with unconsciousness and left-sided convulsions, followed by right hemiplegia. No definite history of fever was obtainable. The child died of broncho-pneumonia about three weeks from the onset of the illness.

Autopsy showed marked cedema of the meninges, with distinct meningo-encephalitis in the region of the central gyri of the left side.

PURVES STEWART.

THE SPINAL CORD AFTER (a) NERVE-CROSSING AND AFTER (346) (b) NERVE-GRAFTING. (*Rückenmarksbefunde (a) nach Nervenkreuzung und (b) nach Nervenpfropfung.*) G. BIKELES, *Neur. Centralbl.*, May 16, 1908.

A SHORT paper in which Prof. Bikeles states the results of nerve-crossing and nerve-grafting in the same animal. In a dog, on the right side the median-ulnar nerve was divided and its peripheral portion grafted on to the intact radial nerve above the branch to the triceps. Eighty-four days later a crossing of median-ulnar and radial nerves on the left side was made, median-ulnar central stump to peripheral radial and *vice versa*. Ten months after the grafting (*i.e.* seven months after the crossing), 6 cm. of each median-ulnar nerve were resected 2 to 3 cm. below the site of graft and cross. One month later the animal was killed and the cord examined (Nissl, *Thionin*). On the left side (crossing) there was degeneration of the cells in the anterior horn corresponding originally to the radial. On the right side (grafting), no pathological alteration of radial cells, but only in the median-ulnar area, *i.e.* that whereas the resection of the median-ulnar on the side of the crossing caused a degeneration of the radial area in the

cord, thus demonstrating the functional continuity of central radial stump and peripheral median-ulnar portion, the lack of changes in the radial area on the side of the grafting shows that the median stump grafted on to the radial nerve never entered into functional union. Where the choice is open, therefore, crossing is to be preferred to grafting.

R. CUNYNGHAM BROWN.

A SIMPLIFICATION OF NISSL'S STAIN AND ITS APPLICATION TO BERI-BERI. (Eine Vereinfachung der Nisslschen Färbung und ihre Anwendung bei Beri-Beri.) ERNST RODENWALDT, *Monatschr. f. Psychiat. u. Neur.*, April 1908, Bd. xxiii., S. 287.

STARTING from Giemsa's view that the active stain produced by the combination of methylene blue and Venetian soap could only be azure, the author suggests the replacement of Nissl's stain by 1:750 watery azure, to which just before use 40 per cent. of saturated calcium carbonate has been added. He stains for only one minute, and without warming. After differentiating in anilin alcohol he passes the section through absolute alcohol, then xylol, origanum oil, canada balsam. (In the reviewer's experience more permanent results may be obtained by using the xylol after the oil and omitting the absolute alcohol stage.)

In a few lines he describes the findings in the spinal cords of two cases of beri-beri. Degeneration was found in the cells of Clarke's column, and of the anterior horn, the antero-medial group being, however, spared. The "fish-eye" stage was that most frequently found.

ERNEST JONES.

PSYCHOLOGY.

EXPERIMENTS TO DETERMINE CO-CONSCIOUS (SUBCONSCIOUS) IDEATION. MORTON PRINCE, *Journ. of Abnorm. Psychol.*, April-May 1908, p. 33.

DR PRINCE is well known as the investigator of the remarkable case of multiple personality known as Miss Beauchamp. The present paper is a description of experimental evidence obtained with a view to deciding between two theories of subconscious manifestations. The first of these—the psychological—regards subconscious manifestations as the expression of subconscious ideas more or less dissociated from the personal consciousness; the second—the physiological—regards them as the result of

physiological processes without any association with ideas whatsoever. The question for Dr Prince was—Could a problem, obviously requiring ideation, be solved by a subconscious (co-conscious) personality while the personality predominant at the time was unaware of the process taking place? He had a case of multiple personality, B-A, in which the one personality, A, was completely unconscious of the other, B, whereas B had full knowledge of A, and claimed also to have a distinct life of her own going on all the time that A was to the fore. The substantiation of this claim would, of course, support the psychological interpretation of the phenomena. The paper contains an account of the tests which Dr Prince imposed upon B, to see whether she could make good her claim. It may be mentioned that she stood the tests, which were chiefly arithmetical in nature, triumphantly.

MARGARET DRUMMOND.

**MEASURE OF INTELLECTUAL FATIGUE IN CHILDREN OF
(349) BOTH SEXES BY MEANS OF THE ÆSTHESIOMETER.**

(Mesure de la Fatigue intellectuelle chez les Enfants des deux Sexes avec l'Esthésiomètre.) SCHUYTEN (Anvers), *Rev. de Psychiat. et de Psychol. Experiment.*, April 1908.

THIS article is to form part of a volume on Feminine Education, which is to appear shortly. It opens with a defence of the æsthesiometer as an indicator of fatigue, a defence supported by special experiments designed to test the question.

Subsequently experiments were performed on school children to determine the annual curve of æsthesiometric fatigue. This is shown to ascend throughout the whole school year, a slight descent marking each of the two holiday periods. To test whether this result was really to be attributed to school work, the author tried a similar series of experiments on young people attending evening classes, and he gives figures to show that no such steadily increasing fatigue is here indicated.

In considering the results obtained, comparisons are made between boys and girls, between the more intelligent and the less intelligent children, and between the right half and the left half of the body.

Accounts are given of æsthesiometric researches with school children as subjects, made by Vannod at Berne and Yasousabouro Sakaki at Tokio.

The writer considers that the results of his work are gravely condemnatory of the whole school system.

MARGARET DRUMMOND.

AN EXPERIMENTAL STUDY OF SLEEP (PART I). BORIS SIDIS,
(350) *Journ. of Abnorm. Psychol.*, April-May 1908, p. 1.

THE writer begins with an account of the various theories of sleep, classifying them under the following headings:—(1) Physiological Theories, subdivided into Mechanical and Chemical; (2) Pathological; (3) Histological; (4) Psychological; (5) Biological. A study of the conditions inducing sleep follows. It is pointed out that these conditions are similar to those relied upon to induce a subconscious and particularly a hypnotic state. The writer finds that subjects whom he is endeavouring to hypnotise frequently pass into ordinary sleep. Limitation of the field of consciousness, limitation of voluntary movement, and, possibly most fundamental of all, monotony, are conditions tending to bring about either sleep or hypnosis, according to predisposition. But while fixation of the attention is an important factor in the induction of the hypnotic state, its relaxation is favourable to the onset of sleep.

Dr Sidis then discusses certain peculiar subwaking states, which he terms hypnoidal; these are unstable in character, passing easily into the ordinary waking state on the one hand, on the other into either sleep or a light hypnosis. They are characterised by a high degree of suggestibility, and by the formation of hallucinations. The method used to induce these states is described.

Guided by the phenomena observed in his study of the hypnoidal states in his human subjects, Dr Sidis determined to carry out a series of experiments on the induction of sleep in animals. The animals employed in this special research were frogs, guinea-pigs, cats, dogs, infants, and adults. This first part of the monograph concludes with an account of the experiments on the frogs and the guinea-pigs.

MARGARET DRUMMOND.

CLINICAL NEUROLOGY.

A CONTRIBUTION TO THE PATHOLOGY OF MYASTHENIA

(351) GRAVIS. Report of a Case with Unusual Form of Thymic Tumour. F. S. MANDLEBAUM and H. L. CELLER, *Journ. of Exper. Med.*, May 1908, p. 308.

FROM a review of the 46 cases which have been published with more or less complete autopsies, the authors conclude that thymic neoplasms have been noted too frequently (11 cases) to be ignored as a possible etiological factor. In their own case an unusual type of tumour, to which they give the name of perilymphatic lymphangio-endothelioma, was present. They consider it probable

that the disease is a manifestation of a toxæmia of indeterminate origin, and that the action of this toxic agency is not confined to the muscular system, but the organism is generally affected as evidenced by the widespread presence of lymphocytic infiltrations (lymphorrhages) throughout the body. They record these latter from various voluntary muscles (very well marked), tongue, liver, adrenals, and some of the perivascular lymphatics of the medulla. No degenerative changes were found in medulla or cord, although the infiltrations would indicate the involvement of the central nervous system in the toxæmia. The change in the muscle fibres are purely degenerative, the result of the toxæmia, and not dependent upon a primary myositis; these degenerative changes bear no relation to the site of the lymphocytic infiltrations.

The article is illustrated by some excellent photographs of lymphorrhages and of the thymic tumour.

J. H. HARVEY PIRIE.

HYPOTONIA. (*Die Hypotonie.*) ALBERT KNAPP, *Monatschr. f. (352) Psychiat. u. Neur.*, Bd. xxiii., *Ergänzungsheft*, S. 16.

IN this monograph, 80 pages long, the whole problem of hypotonia is handled in such a thorough and extensive manner as to render abstraction of the same impossible. First the clinical side is dealt with in detail, stress being especially laid on the diagnostic value of the phenomenon and its independence from other signs which are frequently associated with it. A long and interesting discussion of the theory of the subject follows, in which the author inclines to the pre-Bastian attitude concerning the importance of the peripheral reflex arc. He says, for instance, "The thesis may be laid down with all certainty that up till now no case of complete interruption of the continuity of the spinal cord has been observed in which the loss of muscle-tone and reflexes could not be attributed to anatomical or functional disturbances in the lumbar reflex arc." This, of course, does not exclude action of the higher centres and paths, but injury of these can affect the muscle tonus only *via* indirect injury of the lower reflex arc.

ERNEST JONES.

A TYPICAL FORM OF TABETIC GAIT. (*Eine typische Form der (353) tabischen Gehstörung.*) H. HAENEL, *Deutsche Zeitsch. f. Nervenheilk.*, 1908, Bd. xxxiv., p. 279.

THE gait in tabes is not always of the typical stamping kind, with banging of the heels. Variations in type occur according to the preponderating or exclusive affection of one or another group of

muscles. Haenel describes a common variety of gait which in certain respects may be regarded as typical, and which is useful for the study of normal progression.

For the purpose of walking, in order to advance from the standing posture, the centre of gravity, which lies over a point midway between the two feet, has to be shifted exactly over one foot: only then does the other advancing foot become free to move forward. What is the motor process executed during this shifting of the centre of gravity? A muscle can never "push," it can only "pull"; it exerts power by the process of shortening, not by lengthening. To bend the upper part of the trunk towards the right side in the standing posture, we must first contract some muscle or group of muscles whose fixed point is further to the right than its movable point. The spinal and lateral abdominal muscles, in this respect, are not the efficient group which they might at first sight seem to be, inasmuch as they run more or less parallel with the spinal column and only produce a lateral bending of the vertebræ, not a displacement *en masse*. Inasmuch as lateral movement of the spine upon the pelvis is impossible, the pelvis moves together with the superposed part of the trunk; it has therefore to be approximated to the thigh. This again is impossible, since the neck of the femur prevents any shortening, so that all that the pelvis can do is to change its angle towards the neck of the femur. The fixed point, then, is not the great trochanter, but the foot which is fixed. The chief joints around which rotation occurs are in the region of the ankle joint, and especially the calcaneo-scaphoid joint.

The gastrocnemii, which in the lying position depress the foot, in the erect attitude raise the whole body. The peronei which ordinarily elevate the outer border of the foot, when the border is fixed by the contraction of antagonists and the weight of the body, in the erect posture tend only to draw the upper end of the leg outward. The extended knee-joint permitting of no lateral movement, this movement of the leg is directly transferred to the thigh. With a flexed knee the vastus externus acts as an accessory muscle in the same sense. The hip-joint is capable of movement in many axes, and therefore the pelvis does not follow the thigh directly, but by the help of the short hip-muscles. Now the rotators of the hip pull the pelvis forwards or backwards. The lateral elevators of the hip become lateral depressors, and so on, different muscles coming into action according as the pelvis makes different angles with the femur. The gluteus maximus is specially important for this movement of the pelvis. It is, in fact, the most specifically human muscle we possess, being almost completely absent in the lower animals—even in the anthropoid ape. It is of special significance for the erect attitude. Whereas with most

other muscles the fixed point varies according to the movement performed, with the *gluteus maximus* the fixed point is normally at the femur, preventing the pelvis and trunk from falling forward.

In order to perform the apparently simple movement of lifting one leg, the patient must therefore set a large number of muscles into action, the chief axis of rotation being around the ankle and calcaneo-scaphoid joints. In a tabetic patient in the recumbent posture or with a loosely-hanging leg, we can often detect a distinct pseudo-paresis of the peronei muscles, *i.e.* a drooping and dragging of the outer border of the foot, since there is deficient innervation of the peronei from faulty sensory impressions and later also from diminished muscular tonus. This is shown in an ataxic patient by the tendency for the foot to crumple inward. Such a patient if he tries to lean to the right depresses his shoulder and bends the vertebræ in concave fashion to the right, but the upper end of the right leg tends to move inward instead of outward. Thus he merely rotates his trunk around a sagittal axis passing through his centre of gravity, instead of shifting the centre of gravity to one side as he ought to do. If, therefore, he tries to swing the left leg forwards, the left foot, being not yet unloaded, clings to the ground and requires a great effort to raise it. To correct this, all we require to do is to show the patient that he must incline the fixed leg outwards instead of inwards, when he at once succeeds in overcoming the pseudo-paresis of the peronei.

This deficiency of outward movement of the lower limb is the first of the characteristic abnormalities of the tabetic gait; a second, less commonly recognised, but equally important, is located in the short muscles between thigh and pelvis, especially between trochanter and pelvis. These are the three *glutei* and the *tensor fascia latae*, which act as abductors of the "labile" leg, the *pyriformis*, *obturator internus*, *gemelli*, and *quadratus femoris*, which are outward rotators, and the *obturator externus*, assisted by the *pectineus* and *adductor brevis*, acting as inward rotators. In tabetic patients these muscles become disordered comparatively early in the disease, there is difficulty in holding up the extended limb steadily, and in the lateral position there is special difficulty in raising the upper leg, pseudo-paresis being specially marked. This latter posture is also convenient for demonstrating the different action of the external rotators, by asking the patient to flex his knee, keeping the heels together, and then to raise the upper knee. We also observe well in this posture the hypotonia of the short hip muscles, the great trochanter being unduly prominent, as if the muscles had sagged away from it.

If we examine a tabetic patient in the standing posture, so long as the knees are extended, abnormal lateral movements of the

pelvis are impossible; the most that is seen is a tendency to sway around the vertical axis (less commonly around the frontal axis, since the *gluteus maximus* and *ilio-psoas* are generally least affected). It is quite different when the patient tries to stand on one leg. In health we immediately feel the *gluteus medius* and *tenor fasciæ latae* contract, whilst the pelvis is tilted downwards on the same side and upwards on the opposite side. As a result the sole of the opposite foot is raised from the ground. In a tabetic patient things are quite different. Even supposing he performs properly the outward movement of the fixed leg, yet the short hip muscles do not contract, the pelvis falls over to the opposite side, the stepping lower limb is lengthened instead of shortened, and therefore he has to flex it at the hip and knee. In order to raise the foot from the ground he must bend it further still at these joints. The unsupported leg loads the pelvis still more on that side, the centre of gravity, badly adjusted, tends to fall over, and the patient has to put his foot hastily again to the ground; he cannot lift the leg slowly up and place it down again. He therefore seeks for the support of both feet to maintain his centre of gravity. As soon as the stepping leg touches the ground again it is suddenly extended, the bent knee snaps into the straight posture, and the pelvis becomes again horizontal. This movement goes on alternately with each leg, the limb being placed on the ground bent and then the pelvis brought horizontal by an additional movement, more a passive movement of knee-extension by the quadriceps than of actual gluteal contraction.

Pseudo-paresis of the peronei, though well marked in many cases, can be voluntarily corrected by the patient, if he is shown how to direct his attention to that leg; it may even be masked by compensatory over-action of peronei. Not so with pseudo-paresis of the *gluteus medius*; even by voluntary effort the patient cannot abduct properly the upper leg in the lateral posture.

Haenel then enters into useful details of re-education movements to be practised by tabetics. For successful results personal supervision by the physician is essential. PURVES STEWART.

JUVENILE TABES DORSALIS; Notes of Five Cases. SYDNEY (354) STEPHENSON, *Lancet*, May 16, 1908, p. 1401.

JUVENILE tabes, like the adult form, may assume various clinical guises. One group, in which fall the cases described in this communication, is characterised chiefly by amblyopia due to optic atrophy, together with change in the pupillary reflexes and abolition of the knee and ankle jerks. They correspond to the cases of "pre-ataxic optic atrophy" in the adult, and come, as a rule, first

under the observation of the ophthalmic surgeon. Of the five cases recorded, principally with regard to the ocular manifestations, four are almost certainly cases of tabes, the fifth might be an instance of hereditary cerebellar ataxy.

J. H. HARVEY PIRIE.

THE SYMPTOMS AND DIAGNOSIS OF JUVENILE TABES.

(355) ERNEST JONES, *Brit. Journ. of Children's Diseases*, April 1908.

A REVIEW of our present knowledge of the subject. About thirty undoubted cases are on record. Juvenile differs from adult tabes in the greater frequency of urinary symptoms, of headache and amblyopia, in the slight extent to which girdle pains and ataxy are present. More than a third of the parents of these cases suffered from *metasyphilis*. The differential diagnosis from pseudotabes, hereditary nervous diseases, and cerebro-spinal lues is discussed.

AUTHOR'S ABSTRACT.

A CASE OF INFANTILE PARALYSIS SIMULATING MEN-

(356) INGITIS—TENDON TRANSPLANTATION. A. JEFFERIS
TURNER, *Austral. Med. Gaz.*, March 20, 1908.

A BOY, 3 years of age, took acutely ill in Brisbane at a time when there was a small epidemic of infantile paralysis in that town. He was unconscious for several days, with total flaccid paralysis of the limbs on both sides. He developed a squint and slight head retraction, and for three weeks symptoms of cerebral irritation persisted and increased. The fluid obtained by lumbar puncture was quite clear. Improvement gradually set in, and two months after the onset he was "in fair general condition, but with extensive flaccid paralysis of spinal type." His mental condition was unimpaired.

Two years later the boy was again seen, and it was found that his right arm was his only unparalysed limb.

The left arm was placed in a splint, as was also the left leg, while the transplantation of tendons was done in the right leg with marked success.

A. DINGWALL-FORDYCE.

SUBACUTE COMBINED CORD DEGENERATION, WITH REPORT

(357) OF CASES. J. GRINKER (of Chicago), *Journ. Am. Med. Assn.*,
April 4, 1908.

LICHTHEIM first described the disease in 1887, pointing out the association of pernicious anæmia and spinal cord disease.

Putnam in 1891 showed conclusively that cord lesions are as often associated with other conditions of ill-health with or without anæmia.

Many names have been applied to this disorder—subacute combined sclerosis, subacute ataxic paralysis, combined cord degeneration, diffuse spinal cord degeneration—but the author prefers subacute combined cord degeneration.

The chief symptoms are—(1) sensory, due to posterior tract degeneration, and (2) motor, due to pyramidal tract, and, later, anterior horn degeneration. The symptoms develop somewhat as follows:—Slight impairment of subjective sensation in the legs; tingling, numbness, or prickling in feet, or calves of legs; dull ache in the lower spine; slight inco-ordination in lower extremities; gradually the paræsthesia and inco-ordination extend to the upper extremities. Superficial reflexes usually normal, deep reflexes invariably exaggerated; ankle clonus. Babinski, Oppenheim, and Gordon's reflexes are present. The "spastic ataxia" in the limbs gradually develops and complete paraplegia follows; the cranial nerves, except optic, are rarely involved.

The terminal stage develops in from six months to six years, the deep reflexes are abolished, sphincter paralysis appear, and death from intercurrent disease or exhaustion closes the scene.

Reported cases may be conveniently divided into three groups:—(1) Those in which pernicious anæmia was present from the first; (2) those in which grave anæmia was found associated with subacute combined cord degeneration at some time, but not constituting its most important symptom; (3) those in which anæmia was either slight, absent, or constituted a minor symptom. In 17 cases, 14 were men and 3 women, nearly all were over 40 years, and syphilis had occurred in but one of the 17. Nearly all first exhibited subjective sensory disturbances, disorders of co-ordination, and, later, motor weakness. Only 4 began with typical pernicious anæmia; others showed little anæmia, or, if pernicious, it was not an early symptom. Post-mortem examination showed discrete patches of degeneration in the posterior and lateral columns of the spinal cords, most commonly in the cervical and dorsal regions. In differential diagnosis, spinal syphilis and multiple sclerosis offer the most difficulties; of the etiology little is known, the treatment is entirely symptomatic.

C. H. HOLMES.

FALSE LOCALISATION OF PAIN SENSATION IN A CASE OF (358) COMPRESSION OF THE CORD.

(Über falsche Lokalisation der Schmerzempfindung bei Rückenmarkskompression.)

RENNER, *Deut. Zeitschr. f. Nervenheilk.*, Bd. 34, H. 3 and 4, S. 210.

THE case was that of a man with a tumour compressing the cord at the level of the 7th dorsal vertebra. At the corresponding

level was a band of hyperæsthesia of the skin. All painful stimulation of the legs, genitals, or lower part of the body was referred to this hyperæsthetic zone. Such cases are probably to be explained by irradiation and false projection from the cortex. (For abstract of a similar case reported by M. Lewandowsky, see this *Review* for 1906, p. 703.) J. H. HARVEY PIRIE.

ACUTE PARAPLEGIA FOLLOWING ANTI-RABIC INOCULATION. (359) TION. (Über akute Paraplegien nach Wutschutzimpfungen.)
ED. MÜLLER (Breslau), *Deut. Zeitschr. f. Nervenheilk.*, Bd. 34, H. 3 and 4, S. 252.

In this paper the author adds another to the two dozen cases, which have been recorded, of acute paraplegia following on preventive inoculation for rabies. His case is recorded in detail; an analysis of the previously recorded cases is given; the following is a summary of the facts known about this interesting condition.

There is undoubtedly a typical disease of the nervous system quite different from true human rabies, associated with anti-rabic inoculation; its chief characteristics are acute paraplegia and severe disturbances of micturition and defæcation; the prognosis is very favourable. The site of the disease is chiefly the spinal cord, but the bulbar axis and peripheral nerves may also be affected. Age and sex appear to have no special incidence.

The onset is quite acute—one to two weeks after the first inoculation is the rule. Frequently there are premonitory symptoms indicative of a toxic infectious process (fever, general uneasiness, headache, loss of appetite, pain in back and limbs, etc.); also slight psychical disturbances—nervous unrest and depression.

Along with severe disturbances of the bladder and rectum, there appears at the onset a paresis of the lower limbs, rapidly becoming more and more complete. This is often associated with marked muscular pain and stiffness, especially in the lumbar region. Sensory irritation, in the form of paræsthesiæ or rheumatic, lancinating and neuralgic pains, is also observed.

Usually the paresis of the lower limbs increases in a short time till every trace of voluntary movement is lost in the leg and thigh muscles. Concurrently, there is complete retention of urine and fæces.

Muscular tonus in the lower limbs is usually diminished, and the tendon reflexes are lost. The skin reflexes (abdominal, cremasteric, and plantar) usually also disappear; Babinski's sign may be evoked by strong plantar stimulation. Sensory disturbances vary. Gross disturbances may not be present, but after the original signs of irritation, there may be marked diminution of sensibility, or

even extensive complete segmentary anæsthesia for all forms of sensation; hyperæsthesia of the muscles to pressure may be noted.

In most cases the paralysis is limited to the lower limbs, but not infrequently there follows rapidly paresis of the muscles of the trunk and upper limbs, going on to complete paralysis. Here, also, before the paralysis, there is a feeling of stiffness in the muscles. The process may involve the bulbar region, with development of serious bulbar symptoms. Facial paralysis and paresis of some of the external eye muscles (pupil and fundus being normal) are the most frequent signs, but more serious may be the respiratory and cardiac disturbances (especially tachycardia). Difficulty in swallowing, aphonia, and salivation have been described.

In spite of the rapid development and even appearance of bulbar signs, the prognosis is very favourable; death only rarely follows on long-lasting paraplegia. Usually the remote bulbar symptoms, especially those characteristic of the hydrophobic condition, fail to appear. Above all, there is lacking the peculiar tonic convulsions of true rabies, the spasm of the pharynx and glottis and of the respiratory muscles. Rapid recovery begins after a few days or weeks at most, and is usually complete, though perhaps not for some months. In contrast to poliomyelitis, there is never any atrophic paralysis. Treatment is limited to careful nursing and cessation of the inoculations.

J. H. HARVEY PIRIE.

TWO CASES OF ACUTE MENINGITIS OF THE CONVEXITY.

(360) JOSEPHINE HEMENWAY, *Arch. of Ped.*, March 1908.

CASE I. was a child aged twenty months; the main symptoms were sudden general convulsion, unconsciousness, dilated pupils, slight nystagmus, internal strabismus, temperature 100°, exaggerated knee-jerks, leucocytosis 24,200; no other physical signs were present; lumbar puncture negative; broncho-pneumonia in third week, which cleared up in seven days. Lumbar puncture in fifth week showed increased tension, was otherwise negative. Emaciation; return of signs in lungs; death on seventy-third day.

Autopsy: Meningitis limited to parietal and frontal lobes, superior and lateral aspects; broncho-pneumonia; smears and cultures from meninges and lung both showed Fraenkel's Pneumococcus.

CASE II.—Case showed symptoms similar to those in Case I., and was diagnosed as one of convexity meningitis.

The patient recovered.

Conclusions regarding acute convexity meningitis drawn from these two cases and others observed at the hospital—

(i) Pneumococcal meningitis is usually most marked on the convexity.

(ii) It occurs as a primary condition or secondary, generally to pneumonia.

(iii) There are no symptoms due to basal or spinal lesions.

(iv) Lumbar puncture may be negative.

(v) The nervous symptoms are usually general, and followed by mental symptoms.

JOHN M. DARLING.

EPIDEMIC CEREBRO-SPINAL MENINGITIS IN ADULTS. (Ueber

(361) Meningitis cerebrospinalis epidemica im höheren Lebensalter.)

HERMANN SCHLESINGER, *Wien. Med. Wchenschr.*, April 1908, p. 726.

DR SCHLESINGER makes a few introductory remarks on the rarity of this disease among adults, and old people; he thinks that the atypical course of the disease in these older age limits has not been sufficiently emphasised, and on that account he believes that some of those cases may have been overlooked. Dr Schlesinger goes so far as to say that there is a distinct senile type of the disease, differing from that in children by being more gradual in onset, and he notes particularly the slight degree of rigidity of the neck and its early disappearance.

He does not consider Cheyne-Stokes breathing as being necessarily an unfavourable symptom. There is an interesting series of his own cases in adults, showing how their symptoms varied from the normal type.

Dr Schlesinger lays stress on the importance of the bacteriological examination of the fluid obtained by spinal puncture, and points out the frequent occurrence of cases of tubercular meningitis during epidemics of cerebro-spinal meningitis. The treatment in adults corresponds to that in children.

He lays special stress on repeated spinal puncture, and discusses the value of the different serums in use for the disease.

DUNCAN LORIMER.

PRELIMINARY REPORT OF A CASE OF CEREBRO-SPINAL

(362) MENINGITIS OF STREPTOCOCCUS ORIGIN APPARENTLY CURED BY SUBDURAL INJECTION OF ANTI-STREPTOCOCCUS SERUM.

GEORGE L. PEABODY, *N.Y. Med. Rec.*, March 14, 1908.

PATIENT was a man aged 37 years, and was admitted to hospital on 2nd January, fourteen days after the onset of his illness

Illness commenced with a chill after exposure to cold and wet. His condition on admission suggested cerebral trouble, and his fever was of the type usually found in cases of sepsis. Lumbar puncture on the day after admission showed slightly turbid cerebro-spinal fluid under considerably increased pressure. Culture revealed the presence of a gram-positive coccus of doubtful identity. Lumbar puncture was repeated on 10th January and cultivation showed definite streptococcus after forty-eight hours. On 12th and 13th January 10 c.c. of antistreptococcus serum were injected subcutaneously. No improvement followed. On 14th, 15th, 16th, 17th, 19th and 21st January, 10 c.c. of antistreptococcus serum were injected into the subdural space. After the second subdural injection, the cerebro-spinal fluid was sterile. The temperature fell to normal in eight days after commencement of subdural injections, and remained normal till time of reporting the case (February 17th). Other symptoms also abated, and patient is now apparently quite well. JOHN M. DARLING.

MENINGOCOCCUS HYDROCEPHALUS. FRANCIS HUBER, *Arch. of* (363) *Pediat.*, March 1908.

EFFUSION may take place at any stage in the course of epidemic cerebro-spinal meningitis or meningo-encephalitis. The effusion may be into the sub-arachnoid space—the “external” type; or into one or both ventricles—the “internal” type—or there may be a combination of both varieties.

The amount of enlargement of the head and the shape of the enlarged head are influenced by the degree and extent of sutural union. In the infant, within one or two weeks of the onset, the anterior fontanelle bulges and pulsation diminishes or ceases, the veins of the skull become prominent, and the cranial diameters increase. This is associated with disturbance of nutrition and innervation throughout the rest of the body. In very young infants the increase in the size of the skull is uniform. In older infants the non-united parts of the skull yield most easily. Again, the collection of fluid may be most marked in the anterior cornua, and the eyes may be pushed downwards and forwards; or the posterior cornua may be most affected and give rise to bulging behind with retraction of the head and arching backward of the spine. If the sutures and fontanelles have been obliterated, the head may be normal or very little increased in size.

The clinical manifestations are briefly those:—Within a week or two of the onset of the meningitis, in spite of low or irregular temperature, the patient remains apathetic or comatose, with recurrent vomiting, occasional convulsions, emaciation, retraction

of the abdomen, dilated pupils, distension of the veins of the head and forehead, perhaps strabismus, stiffness and retraction of the head, paroxysmal headaches, hydrocephalic cry, and neuralgic pains in the nerve trunks. The pulse in the young is rapid; in the older it may be slow and irregular. Incontinence of urine and fæces, and muscular paralyses or tremors are frequent. The symptoms often temporarily improve.

Treatment is unsatisfactory, with the possible exception of the iodides. Sometimes the process is cut short by nature. A few grow up with little or no mental change. Cases are recorded where patient was subsequently exceptionally bright. In others reminders occur, such as paroxysmal headaches and pains, or patient may become idiot, epileptic, blind, etc.

As regards operative interference, lumbar puncture gives the best results in external cases. Drainage into the retro-peritoneal tissue has been tried in the combined variety with no ultimate success. In the internal cases, ventricular drainage has been resorted to, but encouraging results are reported in only one case. The results of surgical measures have up till now been unsatisfactory, but earlier diagnosis and interference might improve them.

JOHN M. DARLING.

A CASE OF SYPHILITIC MENINGITIS, WITH AUTOPSY. (Un (364) cas de méningite syphilitique, avec autopsie.) G. BALLET et A. BARBÉ, *Rev. Neurol.*, Ap. 15, 1908, p. 337. (Soc. de Neurol.)

A SERVANT girl, aged 21 years, was admitted to hospital on June 25, 1907, for severe headache. No other nervous symptoms were present, but there was a roseola on the face and trunk. A chancre had not been noticed. Lumbar puncture on June 28th showed an enormous lymphocytosis. Daily injections of biniodide of mercury, associated with the oral administration of 60 grains of potassium iodide, were instituted. It was not till her discharge in the beginning of September, when she had received sixty injections, that the headache and lymphocytosis disappeared. She was re-admitted in October for intense headache, which, in spite of specific treatment, rapidly became worse. Typical symptoms of meningitis developed, and death took place on October 25th.

At the autopsy a lymphocytic infiltration was found in the thickened pia mater. The subarachnoid spaces were also the seat of a diffuse embryonic infiltration. The lymphocytes were most abundant in the neighbourhood of the blood-vessels, which showed evidences of periarteritis and periphlebitis. Points of caseification were found on the lower surface of the hemispheres. Neither the *spirochæta pallida* nor the tubercle bacillus could be detected.

J. D. ROLLESTON.

**AN ATTEMPT TO CLASSIFY CEREBELLAR DISEASE, WITH
(365) A NOTE ON MARIE'S HEREDITARY CEREBELLAR
ATAXIA. GORDON HOLMES, *Brain*, Part cxx., 1907.**

SIXTY-SIX publications referring to cases of primary cerebellar disease are reviewed in this paper with a view to a classification on the basis of morbid anatomy and pathogenesis. The nature and extent of the pathological changes were ascertained by post-mortem examination in each case.

I. *Primary parenchymatous degeneration of the cerebellum.*—Under this head are grouped a number of cases which showed symptoms of a more or less similar nature—reeling gait, incoordination, nystagmus, slow articulation, etc.—and where the main pathological changes found post-mortem were smallness of the cerebellum, as a whole, and atrophy or disappearance of the Purkinje cells, with or without degenerative changes in the molecular and granular layers.

The author considers that the pathological changes which were found by Herringham and Andrews in the cerebella of a litter of four kittens suffering from cerebellar symptoms, and which they regarded as degenerative in origin, were in reality probably developmental abnormalities.

II. *Olivo-ponto-cerebellar atrophy.*—This class includes cases described by Thomas as of “a type characterised *anatomically* by atrophy of the cerebellar cortex, of the bulbar olives, and of the grey matter of the pons; by total degeneration of the middle cerebellar peduncles, by partial degeneration of the corpora restiformia, and by relative integrity of the central nuclei of the cerebellum; *clinically*, by the cerebellar syndrome. It is neither hereditary, familial, nor congenital. It comes on at an advanced age and progresses slowly. It falls into the group of primary cell atrophies.”

A case published by Schweiger where the clinical diagnosis was disseminated sclerosis is regarded by the author as really falling into this class, the spinal lesion being a coincidence only.

III. *Progressive cerebellar disease due to vascular or interstitial changes.*—Four cases are cited under this head in which there were symptoms of cerebellar disease, and where post-mortem examination showed sclerotic and vascular changes in the cerebellum.

IV. *Acute cerebellar lesions.*—This group includes cases of acute ataxia occurring frequently in children during and after infective illness. Histological examination has been possible only at long periods after the onset. The disease is due probably, in some cases, to an acute inflammatory lesion of the nature of encephalitis, and in others to an acute toxic degeneration of the cortical nerve elements.

V. *Degeneration of the spino-cerebellar tracts, the cerebellum being normal or small only.*—In this class of case, and in Class VI., there is no affection of the cerebellum, but they are included here as their most prominent symptoms have been described as those of cerebellar disease. (1) Cases which do not belong to any recognised type. (2) Cases of Friedreich's disease. The author regards the presence of a cerebellar lesion in this disease as an exceptional coincidence.

VI. *Cerebellar symptoms associated with congenital smallness of the central nervous system.*—Falling into this class are the cases of three brothers described by Nonne, and of two brothers described by Miura. In these cases symptoms suggesting cerebellar disease appeared in early manhood. Post-mortem the central nervous system was found to be small, but no histological change could be detected.

Note on "Hereditary Cerebellar Ataxia" of Marie.—No form of disease exists to which the term "hereditary cerebellar ataxia" can be aptly applied. A number of obscure cases, with some symptoms in common, were grouped by Marie and others under this title. Classification must be on the basis of morbid anatomy and pathogenesis. The term should not be retained.

JOHN M. DARLING.

ACUTE BULBAR PARALYSIS WITH AN UNUSUAL SYMPTOM.

(366) A. GORDON (of Phila.), *Med. Rec.*, Feb. 29, 1908.

A WOMAN of 40 years suddenly and without prodromata became unconscious, and remained comatose three days. She regained consciousness, but was totally aphasic, the face was drawn to the right, the speech improved, but never became normal. Five months later a second attack, in which she was not unconscious; the tongue was protruded and caught between set teeth for twenty-four hours; face drawn to the left. Gradual relaxation took place, there remained inability to swallow, to move the tongue, or to masticate; absolute loss of speech and voice; breathing was difficult.

Nearly a year later the following symptoms persist:—She can make only monotonous and identical sounds; no word, syllable, or letter can be uttered; face deviated to the left; left naso-labial fold deepened; tongue tremulous, its movements are limited and reactions of degeneration are present; uvula flaccid; pharynx anæsthetic; swallowing difficult; blowing and whistling impossible; the right facial muscles show partial reactions of degeneration; the grips are normal, gait normal, knee-jerks exaggerated, paradoxical reflex on right side, no Babinski, no sensory dis-

turbances, no eye symptoms. Intelligence well preserved. She understands spoken or written words—no agraphia. Heart shows apical cystolic murmur; no history or evidence of specific disease.

Gordon suggests the following for differential diagnosis:—Acute and chronic bulbar paralysis; pseudo-bulbar palsy; amyotrophic lateral sclerosis with irregular bulbar onset; and hysteria. The latter diagnosis he excludes. He considers the exaggerated knee-jerks, with the presence of paradoxical reflex, in favour of the diagnosis of amyotrophic lateral sclerosis, but seems to consider acute bulbar paralysis the most tangible diagnosis.

C. H. HOLMES.

ADVANCES IN THE DIAGNOSIS OF CEREBRAL TUMOURS.

(367) (*Fortschritte in der Diagnostik der Gehirntumoren.*) KNAPP, *Muench. Med. Wchnschrft.*, May 12 and 19, 1908.

THE surgeon's difficulty in dealing with cerebral tumours depends often not so much on the inaccessibility of the growth as on the impossibility of exactly localising it. Tumours growing in one or other of the well-known cerebral centres give least trouble in diagnosis; these produce direct "focal symptoms" (*Herdsymptome*), and the early surgery of brain tumours confined itself almost exclusively to growths of the cortical motor region.

But removal of a tumour involving, *e.g.*, the motor or sensory speech-centres, usually results in a corresponding aphasia.

This drawback does not, however, apply to operations in the so-called "indifferent" or "mute" (*stumm*) regions of the brain; thus the removal of tumours from these parts is a much more attractive task for the surgeon, and it behoves neurologists to devise means for their diagnosis.

Since focal symptoms are absent in these cases, we must concentrate our attention on the "distal symptoms" (*Fernsymptome*), and try to bring more order into our classification of these.

The author gives instances to show how often what are apparently the most unequivocal focal symptoms may lead us astray in diagnosis.

Although it is by no means clear how the distal symptoms are produced—whether by pressure, displacement, traction, or destruction of neighbouring parts, by disturbance of their blood-supply, or the like—still, the fact remains that the diagnostic significance of these distal symptoms is great, and, in default of a *rationale* of their production, we must base our grouping of them on clinical experience.

The author quotes instances of the successful employment by himself of this empirical method, detailing especially certain phenomena referable to the oculomotor nerve (ptosis, mydriasis, etc.), which occur in tumours of the temporo-sphenoidal lobe.

Another method for localising brain tumours to which he refers is that of *cranial auscultation*, instituted by Phleps. The vertex is struck by a tuning-fork, and, whereas in a normal brain the sound is conducted equally well to both sides of the skull and may be heard with similar clearness over any corresponding points of the two hemispheres, the presence of a tumour at any one point lessens its audibility there; this succeeds best when the tumour is close under the skull-cap, and especially if it be somewhat calcified or otherwise hardened.

Thirdly, the Röntgen rays may be used, but their employment is not often feasible, as there is seldom much difference in consistence between tumour and brain-substance; such growths as can be shown are usually those which have eroded or displaced the cranial bones or have undergone calcification.

The fourth method, which we owe primarily to Neisser, is puncture of the brain through the intact skull, with aspiration. The first puncture determines whether there is actually a tumour in the supposed site, or whether there be some other form of brain-disease. The nature of the growth being ascertained, a series of further punctures is made, in order to discover its exact situation and extent. Brain-puncture is almost painless and can be performed quite easily in the out-patient department: it is only to be carried out, however, when definite localising symptoms have been found on clinical examination. The case is then handed over to the surgeon with an explicit written statement as to the findings.

The surgeon often has great difficulty during the operation in knowing when he has completely extirpated the tumour; to overcome this difficulty the author has a continuous series of small sections from the brain examined microscopically during the operation, and the surgeon continues cutting until the pathologists inform him he has entered healthy tissue on all sides.

ARTHUR J. BROCK.

ACUTE SUPPURATION OF THE SPHENOIDAL SINUSES, WITH (368) INTRACRANIAL AND ORBITAL COMPLICATIONS.

(Akute Keilbeinhöhleneiterung mit intrakranieller und orbitaler Komplikation.) G. TRAUTMANN, *Arch. f. Laryng.*, Bd. xx., Ht. 3, S. 381.

THE patient, a boy æt. 15, was seized with sudden fever, headache, and vomiting; the following day swelling of the right upper lid

was noticed. The mucous membrane of the nose was red, and there was some crusting and mucoid secretion. The fundus of both eyes was normal. Temperature and pulse raised. On the third day the swelling of the lid was more marked, protusion of the eyeball, delirium in the evening, neck stiff and swollen. Diagnosis—probably thrombus of cavernous sinus following on nasal infection. The following day the upper lid of the left eye was also swollen. Lumbar puncture on the fifth day gave a negative result; on ophthalmoscopic examination the veins on each side were dilated and the edge of the disc was slightly blurred. Operation—both frontal sinuses were opened, radical mastoid operation performed on the left side (there was a large dry perforation in the membrane on this side), lateral sinus and dura of the middle cerebral fossa exposed, and cerebrum punctured. Nothing abnormal was found. Death on the eighth day.

Section—empyema of both sphenoidal sinuses, purulent phlebitis of the cavernous sinuses, pus in the left orbit, and to a less extent in the right orbit.

The paper begins with an abstract of previously reported cases.

W. G. PORTER.

REPORT OF A CASE OF CEREBRAL ABSCESS WITH MASKED

(369) **SYMPTOMS.** R. E. COUGHLIN, *N.Y. Med. Journ.*, April 11, 1908, p. 691.

THE case was that of a young woman with a marked family history of tuberculosis; a history of pain in her right ear without any discharge of pus two years previously; a pain in right occipital and parietal region coming on two months before admission to hospital, this pain being constant, unrelieved by treatment, and worse at night; forcible vomiting; vertigo; leucocytosis of 18,000; low tension pulse; death without location of her disease. The necropsy revealed an irregular burrowing abscess in the left hemisphere just external to the lateral ventricle. The burrowing pus could be traced to a small opening entering the right ear.

J. H. HARVEY PIRIE.

DISSEMINATED SCLEROSIS COMMENCING WITH FAILURE

(370) **OF VISION.** R. T. WILLIAMSON, *Lancet*, May 2, 1908.

IN this article a description is given of a form of disseminated sclerosis which commences with failure of vision. A long period, often months, or even years, may elapse before other symptoms of

the disease develop. The visual failure is due to a patch of sclerosis in the optic nerve or chiasma. Frequently after the visual defect has become marked there is decided improvement; in other cases the visual failure persists, and partial or marked optic atrophy may develop. At the early stage there is often pallor of the temporal half of the optic discs, with a central scotoma in some cases. At a later period other signs in favour of disseminated sclerosis may be detected, such as the extensor type of the plantar reflex, the irregular and shaky character of the handwriting, even when tremor of the hand on movement can hardly be detected. The patients are usually under forty years of age. In course of time the characteristic symptoms of disseminated sclerosis may gradually develop. Short notes of cases of this form of the disease are given. A micro-photograph of a section of the optic nerve and a drawing of a section of the optic chiasma, from one of the cases, are reproduced in the article. In this case there was a patch of sclerosis in the optic chiasma, which extended into the optic nerve.

AUTHOR'S ABSTRACT.

A CASE OF JACKSONIAN EPILEPSY, WITH AUTOPSY. (Su (371) di un caso di epilessia jacksoniana, con autopsia.) G. SANNA SALARIS, *Riv. Ital. di Neuropat. Psichiat. ed Elettrotet.*, May 1908.

THE case is that of a man, 28 years of age, having a syphilitic history, who developed attacks of local epilepsy, involving the left side, and leaving a hemiparesis in their wake; they occasionally became generalised, in which case consciousness was lost, though this did not occur when the convulsions remained unilateral. Patient died in the *status epilepticus*.

The autopsy revealed a whitish, leathery gumma, of the size of a pea, situate in the fissure of Rolando on the right side, and attached to the pia mater. In the sub-cortical substance immediately below the gumma was an area of softening, of the size of a pigeon's egg, surrounded by a capsule of fibrous tissue.

This tumour had probably originated in a specific inflammation of one of the coats of an arteriole; in its growth it had finally obliterated the vessel, thereby bringing about softening of the area supplied by it. The first convulsive manifestation had probably been due, not to direct irritation of the cortical motor centres by the gumma, but rather to irritation depending on the partially obstructed blood-supply. The gumma itself was too small to have caused the hemiparesis directly; this must have been due to the larger subjacent focus of softening, which involved considerably more of the corresponding motor fibres.

The local epilepsy might conceivably depend either on direct cortical irritation by the gumma, or on the circulatory disturbances produced by it in the brain; it is known that epileptic convulsions confined to certain muscular groups, or even when generalised, may be related to alterations in the circulation or chemism of the brain produced by tumours which are situated in parts more or less distant from the Rolandic motor areas.

ARTHUR J. BROCK.

TROPHIC DISORDERS IN HYSTERIA. (Contribution à l'étude (372) des troubles trophiques dits "hystériques.") A. M. BONO (de Rome), *Tribune médicale*, April 11, 1908, p. 213.

THIS paper, which was inspired by Babinski, is based on observations made in the Paris hospitals during a period of seven months. Among the large number of hysterical patients who had come under his observation Bono had not met with a single case of trophic disorder, *e.g.*, pemphigus, ulceration, or gangrene, of fever, of hæmatemesis or hæmoptysis, which could be undoubtedly attributed to hysteria. The writer thinks that the attribution of such symptoms to hysteria is the result of an error in diagnosis, an error due either to defective observation on the part of the physician or to simulation on the part of the patient. Numerous illustrative examples are given. Bono also addressed inquiries to many of the leading medical men in Paris as to whether they had seen undoubted cases. The almost invariable reply was that they had not seen a single definite case of hysteria which proved the existence of these disorders. Among neurologists who answered to this effect were Gilbert Ballet, Brissaud, Dupré, Landouzy, Pierre Marie, and Sicard, and among dermatologists Brocq, Darier, Hallopeau, Jacquet, and Thibierge. Bono concludes that if such disorders exist they must be so exceptional that they need not be taken into account.

J. D. ROLLESTON.

THE CLINICAL VALUE OF THE DISSOCIATION OF THE (373) CUTANEOUS AND TENDON REFLEXES IN HYSTERIA. (Essai sur la valeur clinique de la dissociation des réflexes cutanés et tendineux dans l'hystérie.) A. FAYET, *Thèses de Lyon*, 1906-1907, No. 39.

DISSOCIATION of the patellar and plantar reflexes, *i.e.* exaggeration of the knee jerk and diminution or abolition of the plantar reflex, is very frequent in hysteria, and should be regarded as a stigma of this neurosis. The centres for these reflexes are distinct, the

centre for the knee jerk being situated in the mesencephalon and that for the plantar reflex in the cortex. The exaggeration of the patellar reflex is attributed to the removal of the inhibitory action of the cortex, the functional disturbance of which in hysteria is also the cause of the suppression of the plantar reflex.

J. D. ROLLESTON.

FREUD'S THEORY OF HYSTERIA. (*Die Freudsche Hysterie-theorie.*) C. G. JUNG, *Monatschr. f. Psychiat. u. Neur.*, April 1908, Bd. xxiii., S. 310.

THIS paper, which was read before the International Congress in Amsterdam, suffers under the obvious disadvantage that it attempts to codify Freud's scattered contributions to the theory of hysteria, while Freud himself has refrained from such a task. Jung adopts the historical method, tracing the development of Freud's views from the first paper in 1893, in which was established the fact that hysterical symptoms are individually traced to a previously experienced psychical trauma. Underlying each symptom, therefore, is a disagreeable mental "complex" (feeling-invested idea) which is repressed (*verdrängt*) and split off from the patient's main stream of consciousness. The stimulus arising from such complexes is "converted" into various symptoms, bodily and mental. The mechanisms by which such conversion occurs have been closely analysed by Freud, who showed that resuscitation of the repressed memory was followed by disappearance of the corresponding symptom. In later years Freud has gone deeper into the study of the origin and formation of such complexes, and sees in precocious sexual excitement, the memory of which has been repressed, and which is often of a perverse character, the necessary basis on which they develop. As the paper itself is an extremely condensed account of Freud's views, and is of little use in replacing a study of the original contributions, it does not lend itself to further abstraction.

ERNEST JONES.

POST-TRAUMATIC, TRANSITORY DISTURBANCES OF CONSCIOUSNESS. (*Posttraumatische, transitorische Bewusstseinsstörungen.*) KARL WENDENBURG, *Monatschr. f. Psychiat. u. Neur.*, Bd. xxiii., *Ergänzungsheft*, S. 223.

THE main thesis of this article is that poriomania may be caused not only by epilepsy, hysteria, and dysphoric degenerative conditions, but also by traumatic neurasthenia. Besides discussing

similar cases recorded in the literature, the author relates the case of a student, aged 25, who, after a fall that was followed by unconsciousness for three hours and was alleged to cause a fractured skull—the evidence for this is most inconclusive—suffered from a number of symptoms usually grouped as neurasthenic. In addition there was marked continuous and anterograde amnesia, which sometimes caused him to forget many hours of his life at a time. This last symptom points strongly to the hysterical nature of the case—though no physical signs of hysteria were present—but unfortunately no psycho-analysis was made. Two months after the accident the patient found himself in a train in the east of Italy with evidence in his pockets showing that he had been travelling for three days in a secondary state. The symptomatology and diagnosis of such cases is discussed, and the author comes to the conclusion mentioned in the first sentence above.

ERNEST JONES.

SPEECH DISTURBANCES IN FUNCTIONAL PSYCHOSES.

(376) (*Sprachstörungen bei funktionellen Psychosen.*) ALBERT KNAPP, *Monatschr. f. Psychiat. u. Neur.*, Bd. xxiii., *Ergänzungsheft*, S. 97-124.

AFTER a few words on the commoner—aphasic, etc.—speech disturbances met with in the psychoses, Knapp discusses four groups of such disturbances as have not hitherto been described in this connection. Thirteen personally studied cases are described in detail. The first group is that of stammering (Stottern), which could not be distinguished clinically from usual stammering except by its transitory occurrence. The cases were of diverse nature, but all showed abnormal psychomotor phenomena, and Knapp regards the stammering thus as being also a parakinesis. The same applies to the second group—scanning speech—which was most often combined with the first set of disturbances. The third and most important group was that of syllable-stammering (Silbenstolpern), which was very similar to the speech change found in general paralysis. The fourth group was that of infantile speech in which blesitas—replacement of one consonant by another—occurred (Stammeln).

ERNEST JONES.

HEADACHE AND EYE-STRAIN. WM. CRAWFORD (Hamilton, Ont.), (377) *Canadian Practitioner and Rev.*, March 1908, p. 155.

THE author discusses the varieties of headache that may be brought about as the result of eye-strain. The first and most frequent variety is brow-ache or supra-orbital headache over one or both

eyes. The next is that of deep orbital site, the pain being concentrated in the eyeball. This occurs most frequently where the defect is astigmatism. The fronto-occipital is another form, and may be most acute in the morning, following on a previous day's eye-strain, particularly that due to astigmatism with axes deviating from the vertical.

To distinguish a headache due to eye-strain from one brought about by other causes, it is the author's opinion that a fairly safe rule is to keep in mind the fact that eye-strain headache is aggravated by eye-work and lessened or relieved by rest. The headache may or may not be accompanied by asthenopia. The smaller ocular defects, such as hyperopic astigmatism of one-half to three-quarters of a diopter, or a like amount of mixed astigmatism, give rise to the most troublesome headaches, especially if the axis be at an angle or against the rule. And here the vision may be nearly normal. The causes of asthenopia are disordered innervation, caused by errors of refraction, and anomalies of the extra-ocular muscles, and are also due reflexly to nasal obstruction, dental caries, and other nose and throat affections. The larger refractive defects usually result in suppression of the visual image or in squint. The eye defects may be unsuspected, and often go for years uncorrected, especially in children. Many cases of congenital astigmatism do not suffer from headache until they reach adult age, when the condition may manifest itself. Uncorrected refractive errors and muscular anomalies are prolific causes of disease conditions in the eyeball itself. Very many grave conditions, such as retinitis, chronic iritis, etc., may be produced or aggravated by uncorrected ocular or muscular defects. The improvement in the general health brought about by the correction of an ocular defect is dwelt on briefly. The author believes that cases of epilepsy have been cured by like treatment, and that the symptom of vertigo is often relieved in this way. Cases of gastric disturbance are believed to have been due to ocular defects, the correction of which led to improvement in the gastric condition. The lowering of general body-tone due to eye-strain is thought to have a distinctly deleterious effect in many cases by bringing about a condition of lessened resistance on the part of the organism. Four cases are given in abstract showing the beneficial results accruing from the correction of ocular defects. The author believes that 50 per cent. to 80 per cent. of cases seen in ophthalmic practice suffer from headache. Most eyes have a slight refractive error, but only in those cases where the eyes are overworked, or the patient is below par, is correction needed. Some such patients require only temporary correction during the time that the eyes are being given an unusual amount of work. The comparative frequency of eye troubles in school children is

commented on, and it is noted that hyperopic errors greatly outnumber emmetropic and myopic conditions in children. Myopia, however, which is comparatively rare before the beginning of the educational process, advances steadily in percentage with the progress of the pupil in the school. The importance of the eye examination in school children is next touched on. The early presbyopes form a class which is often overlooked, and their troubles are readily corrected by the use of a weak lens for close work. Many people who have reflex symptoms of eye-strain such as headache or nervousness rarely suffer from local conditions of the eyeball, such as blepharitis, etc. Anomalies of the extra-ocular muscles are a fruitful cause of eye-strain, and can often be corrected by proper treatment directed to the refractive error. "The demands of modern business, the struggle for existence, make demands on no other organ of the body to so great an extent as on the organs of vision, and they in turn demand from the general system a large amount of nerve force."

FITZGERALD.

SCIATICA. M. L. BARSHINGER, *N.Y. Med. Rec.*, April 25, 1908.
(378)

THE favourite seat of sciatica to begin with is the sciatic notch. Now the sciatic nerve leaves the pelvis through the sciatic notch lying on the bone in front, and with the piriformis muscle resting on it behind. With a history of rheumatism, gout, or exposure and pain referred to the sciatic notch, it is reasonable to presume that the muscle is the seat of a local irritation, as happens in other parts of the body. The piriformis muscle then presses on the sciatic nerve owing to contraction following the irritation, and, in addition, the congestion and swelling of the muscle nips the nerve as it passes through the unyielding constriction. The piriformis may be affected only secondarily by extension from adjacent muscular structures. Women suffer from true sciatica less than men, owing to the structure of their pelvis. The nerve becomes secondarily affected by continued pressure.

The aim of treatment, therefore, should be to free the nerve as soon as possible from compression. This may be done by means of mechanical vibration carried out until the muscle is quite released, *i.e.* till the pain disappears. Radiant energy applied by means of a high-power lamp—for example, the leucodescent—increases the supply of blood to the part, and facilitates the removal of irritating material. Static electricity—the wave current followed by sparks—is very effective. Galvanism and the Roentgen rays are also of use.

JOHN M. DARLING.

ADIPOSIS DOLOROSA. JULE B. FRANKENHEIMER, *Jour. Amer. Med. (379) Assoc.*, March 28, 1908.

DR FRANKENHEIMER gives a full report of a typical case of this disease, which improved considerably under treatment with thyroid tablets. He then proceeds to a general consideration of the recorded cases (about 45).

Etiology.—Cases have occurred in different members of the same family. Women are more commonly affected than men, in the ratio of six to one. The age incidence is variable. In men the majority of cases occur between the ages of 30 and 40, and in women between 30 and 50. Neuropathic and alcoholic histories, syphilis and trauma were noted in a few cases. Disturbance of the sexual organs was frequently found. The menopause seems to predispose to the disease.

Pathology.—Notes of five autopsies only were available. The fatty tissue was of normal structure. One case showed interstitial neuritis and sclerosis of the columns of Goll (probably a concomitant affection). The thyroid gland was enlarged and calcareously infiltrated in two cases, partially atrophied in two cases, and normal in one case. The pituitary body showed enlargement and gliomatous degeneration in two cases, and adeno-carcinoma in one case.

Clinical Manifestations.—There are four cardinal symptoms, viz.:—

1. Accumulation of fat, usually localised.
2. Pain and tenderness in the fatty swellings.
3. Asthenia.
4. Psychic disturbance (apathy and depression, irritability, dementia).

Other symptoms are headache, disturbance of sensation, vasomotor phenomena (lessened perspiration, cyanosis, ecchymosis, trophic ulcers, etc.). Death is usually due to some intercurrent affection. Exacerbations are common.

Conclusion.—The sex incidence is practically the same as that of exophthalmic goitre and myxoedema. The involvement of the thyroid gland is demonstrated by the pathological findings, the presence of myxoedematous symptoms and the positive therapeutic results of treatment with thyroid extract. Further study is necessary to prove that disturbance of the genital organs is a factor in the production of the disease.

It is suggested that "adiposis dolorosa, like acromegaly, is a dystrophy, the one affecting the fatty, the other the osseous structures; and that there is present a toxæmia dependent on a dysthyroidismus and the disturbed function of the pituitary body and the genital organs."

JOHN M. DARLING.

**THE TOE-REFLEX (A SPECIAL PATHOLOGICAL TENDON-
(380) REFLEX).** [Der Zehenreflex (ein speziell pathologischer
Sehnenreflex.)] G. J. ROSSOLIMO, *Neur. Centralbl.*, No. 10,
1908.

FIVE years ago Rossolimo published an investigation containing a description of a new reflex, which he named the deep reflex of the great toe. It takes the form of a bending downwards of the great toe on stimulation of the plantar surface, and is seen in cases of disease of the pyramidal tracts of the cord. It was found to be present in many cases when Babinski's sign could not be elicited. Experience at that time showed that it was a more certain index of organic cord disease, and further investigation, the results of which are given in this paper, confirms this.

This second paper states the results arrived at from an investigation of 91 cases of organic disease of the central nervous system with affection of the pyramidal tracts. The duration of the disease, the condition of the motor functions, the muscular tonus, and especially the state of the various known reflexes, were all carefully studied. To elicit the particular reflex he draws attention to, the finger is gently stroked on the plantar surface of the toe, and after a longer or shorter time a muscular contraction is observed causing flexion or abduction of the toe. The patient may be sitting or lying with the knee bent and the muscles relaxed. There are variations and degrees of the reflex. The great toe, the other four toes, or all five, may show movement, the last being the most common. The spontaneous movements of the toes, the muscular tonus, the condition of the other reflexes, and the time after the onset of disease at which the reflex makes its appearance, were all carefully observed. The author comes to the following conclusions:—

1. The toe-reflex is observed only in affections of the pyramidal tracts, unlike many other pathognomonic reflexes (Babinski's sign, increased patellar and Achilles tendon reflexes) which are also occasionally present in the neuroses.

2. It can be seen in many cases in which Babinski's sign is absent.

3. Its intensity is variable, from hardly perceptible bending to marked clonus.

4. Unlike other tendon reflexes, whose pathognomic value lies in the increase of reflexes normally present, the toe-reflex, like Babinski's sign, is pathognomic in its mere presence, as it is absent normally.

5. Owing to its rare appearance before the disease of the pyramidal tracts has been in existence three weeks, it is a more distinctive sign than Babinski's.

JAMES MIDDLEMASS.

PARATHYREOGENIC LARYNGOSPASM. (Ueber Parathyreogenen (381) Laryngospasmus.) FRIEDRICH PINELES, *Wien. Klin. Wochenschr.*, Nr. 18, 1908.

THE writer states that glottic spasm is due to irritation of all the recurrent laryngeal fibres with preponderance of the closers of the glottis. The causes may be stated as follows:—

1. Diseases of the larynx—severe catarrh, foreign bodies, polypi.
2. Tumours in the neighbourhood of the larynx, aortic aneurysm, mediastinal growths, caseated bronchial glands.
3. Reflex causes from nose, alimentary canal and genital organs.
4. Neuroses—hysteria, neurasthenia, tetany.

It is easy to understand the presence of glottic spasm in cases of tetany, as, in this condition, we have to deal with a state of general muscular spasm, and, in the tetany of children, glottic spasm forms the most striking feature of the disease. Frankl-Hochwart has only recorded 8 cases of laryngeal spasm out of 122 cases of tetany among adults, and states that the condition is rare in them as compared with children. Pineles has come to the conclusion that all human tetany is caused by the same poison—the “parathyreopraver” tetany poison—which, after removal of the parathyroid glands, develops its injurious influence on the organism. Pineles has found records of only two cases of glottic spasm in patients whose thyroid glands have been removed—the cases of Eiselsberg and Dienst; in the latter the patient was pregnant. Laryngospasm was present in all of the four cases of thyroidectomy observed by Pineles himself, and he therefore concludes that this symptom has been overlooked by other observers: in two out of the four cases glottic spasm remained after the other symptoms of tetany had passed off, thus closely resembling the condition in infantile tetany. Under the heading “Parathyreogener Laryngospasmus” Pineles records two cases of Hoffmann’s and four new cases of his own—thus bringing up the number of his own observations to nineteen. Case 3, a female, aged 32, had a child which also suffered from glottic spasm. The author considers that glottic spasm is much more common in idiopathic (parathyreogenic) tetany than would appear in the literature of the subject; and further holds the view that “parathyreopraver” and “parathyreogener” tetany are so much alike that there can be no doubt as to the common origin of the two conditions. He gives two cases of tetany in children in which the mothers also suffered from this condition—one of the mothers

having glottic spasm. It has been proved experimentally in animals that the larynx of the young reacts more quickly and intensely to the "parathyreopraver" tetany poison than that of older animals; also that section of both recurrent nerves produces more severe dyspnoea in the young than in the old. Children are also more affected by double paralysis of the glottis-openers than are adults. These facts explain why the "parathyreopraver" tetany poison produces glottic spasm more frequently in children than in adults.

J. S. FRASER.

INJURIES OF THE VAGUS NERVE AND THEIR CONSEQUENCES. (382) **QUENCES.** (*Die Verletzungen des Nervus vagus und ihre Folgen.*) H. REICH (of Tübingen), *Beitr. zur klin. Chir.*, Bd. lvi., H. 3, 1908.

THE author first discusses accidental injuries of the vagus nerve, of which he has gathered eleven cases. In all the cases severe respiratory disorders were observed. The descriptions given by the author are frequently obscure and leave in doubt whether the symptoms are due to direct involvement of the vagus (impairment of the sensibility of the lung and subsequent pneumonia), or to difficulty of breathing owing to hæmatomata or inflammatory processes in the region of the trachea. The second part of the communication is more important, and deals with sections and resections of the vagus (forty-four cases). Most of the cases were of tumours, usually malignant, which were either intimately connected with the vagus anatomically or had caused its degeneration, so that complete extirpation of the tumour was only possible with resection of the vagus, either intentional or unintentional; in no case was the section bilateral.

The clinical results of such operative interference with the vagus are divided by the author into immediate and remote. Only in five cases were there any irritative cardiac symptoms; these always consisted of tachycardia, lasting from a few hours to fourteen days. The tachycardia was never of serious import. Similarly, the author attributes little importance to the few disturbances of breathing which are described. The pneumonias, which occur later, are demonstrated by the author by means of fully analysed case-histories to stand in indirect ætiological relation to the lesion of the vagus. Paralysis of the vocal cords due to paralysis of the recurrent is always present on the operated side. The author does not refer the mild disorders of the gastrointestinal tract to lesions of the vagus. In his discussion, however, he leaves it an open question how far the primary disease had already caused a slow degeneration of the nerve, and comes

to the logical conclusion that the onset of vagus symptoms presupposes the integrity of the nerve. The author does not consider that there is any mortality associated with unilateral section of the nerve. It is different with irritation of the vagus. One frequently meets here symptoms varying from slight lowering of the blood pressure to momentary asystole with severe symptoms and even with fatal result. More frequent, and as a rule more severe, are symptoms due to the influence of irritation of the nerve on the respiration, which frequently comes on abruptly and may lead to the most severe asphyxia. Naturally the irritative symptoms are most marked when heart and lung are simultaneously affected. The above interpretation of the clinical phenomena is confirmed by experiments on animals.

The main results of this thorough work can be briefly summarised. Section of the vagus without irritation is not dangerous; there only remains permanent paralysis of the vocal cords. The cardiac symptoms are quite unimportant; the symptoms of the other organs are not to be referred to the section of the vagus. If the vagus be accidentally cut, an attempt to suture it should be made. In contrast with section of the vagus, traumatic irritation of the vagus is followed by most severe cardiac and pulmonary symptoms. Animal experiments confirm this view. Irritation of the vagus can kill by stopping the action of the heart and lung. The other conclusions belong to clinical surgery.

GOLDSTEIN (C.g.B.).

A CASE OF INTERMITTENT EXOPHTHALMOS. (Ein fall von (383) intermittierendem Exophthalmus.) MAX MEISSNER, *Med. Blätter*, April 25, 1908, S. 193.

THIS unusual condition, of which some forty cases are on record, was observed in a man of twenty-four, who, for about five months, complained that on stooping his right eye "fell out." At first, this only happened on severe exertion, latterly it had become more frequent. Under ordinary circumstances no difference could be made out between the two eyes, and both appeared sound in every way. When he stooped for a couple of minutes, there was slight swelling and redness of the lids and marked proptosis of the right eye. On resuming the upright position, the exophthalmos disappeared completely in about one minute. When present, vision was blurred, and ophthalmoscopically there was marked venous pulsation. Similar proptosis could be produced by forcible expiration with nose and mouth closed, by manual compression of the right jugular vein, and to a less degree by forcibly turning the head to the right. For some time forcible

pulsation could be felt in the eyeball when protruded, latterly this had disappeared. No cause could be found, and the pathogenesis of the condition seems to be as uncertain as when first described by Sattler in 1880.

J. H. HARVEY PIRIE.

ON THE MORE RECENT RESULTS OF INVESTIGATION IN
 (384) **SYMPATHETIC OPHTHALMIA.** (Ueber neuere Untersuchungsergebnisse bei der sympathischer Ophthalmie.) GEORG
 LENZ, *Berl. Klin. Wchnsch.*, April 27.

IN regard to the important question whether any special signs may be recognised in the primarily affected eye which would lead us to expect a sympathetic inflammation in its fellow, the author holds that such a specific anatomical picture does actually exist. The pathogenic agent he believes to be a specific micro-organism which passes from one eye to the other by way of the general blood-current; it confines its attacks to the uveal tract, just as the tetanus bacillus does to the nervous system.

This theory as to the mode of transmission of the *materies morbi* from eye to eye involves abandonment of the theory that it passes by the optic nerve, as also the "ciliary-nerve theory" of Schmidt-Rimpler, according to which sympathetic inflammation is set up by an irritation conveyed through the ciliary nerves.

ARTHUR J. BROCK.

REFLEX NYSTAGMUS INDUCED AS A MEANS OF DIAGNOS-
 (385) **ING THE FUNCTIONAL CONDITION OF THE VESTI-**
BULAR APPARATUS. (Le Nystagmus réflexe provoqué comme méthode de diagnostic des états fonctionnelle de l'appareil vestibulaire.) E. LOMBARD and E. HALPHEN,
Prog. Med., 3^e Serie, T. xxiii., No. 16.

THE nystagmus discussed in this paper is of vestibular origin. The amplitude of the nystagmus is markedly increased if the eyes are turned to the side to which the nystagmus is directed. The direction of the nystagmus may be horizontal, vertical, or rotary.

A spontaneous nystagmus occurs, it may be observed, where the labyrinth is intact, and it is present in nearly all the acute affections of the labyrinth. Induced nystagmus is much more valuable as a diagnostic sign, it can be set up by rotation, by the application of the galvanic current, of compressed air and of heat and cold within the meatus.

If the endolymph in a canal is made to flow towards the

ampulla, nystagmus is induced in the opposite sense. Hence if the subject be rotated to the right, nystagmus will occur to the right during rotation and to the left after; but if the labyrinth is diseased, the nystagmus may not appear, or may be modified.

If cold water is injected in the right ear of a normal subject with head erect, rotatory nystagmus appears to the left; with hot water the direction is to the right; but if the head be bent to the opposite shoulder, injection of cold water induces horizontal nystagmus to the right.

A number of illustrative cases conclude the paper.

W. G. PORTER.

AMBLYOPIA FOLLOWING ON ACCESSORY SINUS SUPPURATION. (386) **TION.** (*Amblyopie infolge von Nebenhöhleneiterungen der Nase.*) RÉTHI, *Wien. Med. Wchnschr.*, 1908, Bd. lviii, S. 1066.

THE patient, a male æt. 39, had had for three years a tendency to colds in the head. A year ago polypi were removed from both sides of the nose. Six months later he began to suffer from pain in the forehead and temples and from failing sight in the left eye. This gradually got worse. The temporal side of the field of vision was lost, and the sight in the right eye also began to fail. Ophthalmoscopic examination showed bilateral optic neuritis, retrobulbar, probably due to ethmoidal suppuration. The middle turbinal was removed, and the ethmoid labyrinth was cleared out on both sides. The sphenoidal sinuses appeared normal. Four weeks later there was marked improvement of vision. Réthi draws attention to the importance of cases such as this, where there is no swelling of the lids or exophthalmos to point to the nasal cause of the condition.

W. G. PORTER.

ROUSSEAU'S DISEASE. (*La maladie de Jean Jacques Rousseau.*) (387) PONCET et LERICHE, *Bull. de l'Acad. de Méd.*, 1907, p. 607.
CALVIN'S DISEASE. (*La maladie de Calvin.*) PONCET et LERICHE, (388) *Lyon médical*, 1908, p. 801.

IN these studies in pathography the writers have done good service in substituting for the vague term neuro-arthritis a more definite conception of the infirmities from which Rousseau and Calvin suffered.

In a recently discovered testament of Rousseau a complete description of all his symptoms is given, permitting of a precise diagnosis of his disease, which the writers hold to be congenital stenosis of the bulbo-membranous portion of the urethra. There

has hitherto been too great a tendency to attribute Rousseau's urinary troubles to his mental condition. Instead of considering him as "a psychasthenic with his bladder in his head," the writers think that many of his eccentricities were aggravated, if not actually produced, by a prolonged dysuria. Rousseau undoubtedly was neurasthenic and suffered from obsessions, and it is very probable that he had arterio-sclerosis and was prematurely senile, but Poncet and Leriche think that the condition of his urinary passages fully accounts for all his morbid symptoms. In the subsequent discussion Landouzy stated that the neuro-arthritis and neurasthenia of Montaigne were secondary to renal lithiasis. Neuropathologists, he thought, were too apt to be satisfied with the diagnosis of neurasthenia without searching for the causal disease, which remained in the background often in a larval or atypical form.

Tuberculosis of insidious evolution and protracted course, but relatively benign in its local manifestations, played the chief part in the pathological history of Calvin. Overworked, badly nourished, and living in deplorable hygienic surroundings, Calvin suffered from an early age from repeated colds and violent migraine. Subsequently he developed pleurisy and hæmoptysis. On several occasions he had severe colic and diarrhoea, which may have been due to tuberculous ulceration of the intestines. Three years before his death, which occurred at the age of fifty-five, he suffered from gout and nephrolithiasis. Hæmorrhoids, pruritus ani, perineal eczema, and anal fistula added to his distress, which was aggravated by the calumnious interpretation which his enemies made of these local phenomena. J. D. ROLLESTON.

PSYCHIATRY.

CLINICAL AND ÆTIOLOGICAL ASPECTS OF "ZWANG"

(389) PHENOMENA. (Zur Klinik und Aetiologie der Zwangsercheinungen, über Zwangshallucinationen und über die Beziehungen der Zwangsvorstellungen zur Hysterie.) R. THOMSEN, *Arch. f. Psychiat. u. Nervenkrank.*, Bd. xliv., S. 1-5.

THE author first details eleven cases, giving, however, no analysis of the psychological processes concerned. He defines a "Zwangsvorstellung" in Westphal's original sense as an idea which forces itself on consciousness, and which is not conditioned by any affect (!). In contradiction to Janet, Loewenfeld, and others, he emphasises the intimate relation of such phenomena to hysteria

and considers that the majority of the cases belong here. He takes no account of the modern work done on the subject.

ERNEST JONES.

THE PSYCHOSES OF INFLUENZA. R. DODS BROWN, *Scot. Med. (390) and Surg. Jour.*, June 1908, p. 509.

THE author is of opinion that mental disorder comes on more frequently after influenza than after any other febrile affection.

Pfeiffer's bacillus or its toxin seems to be the exciting agent rather than the fever or the post-febrile exhaustion, because the mental derangement may appear not only after severe cases of influenza, but also after those of a mild type, and in those patients whose illness has been of short duration. Moreover, it is found that the mental symptoms may set in as early as the second or third day of the disease.

A bacteriological examination was made of the cerebro-spinal fluid of three patients suffering from acute mania, but in no case was the influenza bacillus isolated. No apparent effect was produced by inoculating mice with the same fluid.

Almost any form of psychosis may occur after influenza, but mental depression is what is most frequently seen.

Contrary to what is usually found, melancholia was not in excess of other forms of psychosis in the writer's series of twenty cases. These consisted of fifteen women and five men. There were ten cases suffering from mental depression, five of whom exhibited great agitation, restlessness and restiveness. Of the remaining ten, nine were classified as cases of acute mania, and in these there was complete or almost complete dissolution of mind with hallucinations of sight and hearing, and in seven of them there was great restlessness and violence.

The tenth case in the manic group was one of delusional mania. The small percentage of melancholics in this series is probably due to the relatively large number of female patients.

Associated with the depression there is very often a strong suicidal tendency.

A noteworthy feature is that the most severe forms of mental derangement after influenza occur in those patients with a predisposition to insanity.

Although there is a great liability to relapse in all these cases, the prognosis as to recovery is good. AUTHOR'S ABSTRACT.

ADOLESCENT INSANITY. (*Jugendirresein.*) KOICHI MIYAKE
(391) (Tokio), *Arb. a. d. Neurol. Instit. a. d. Wien. Univ.*, Bd. xvi.,
Teil 1, 1907.

THIS paper is preliminary to a detailed work on the subject, and the material was obtained during three years' experience in the Clinique of Psychiatry at Tokio. The classification used is that of Kraepelin.

Dr Miyake has observed 1733 cases of insanity. Of this number 8·5 per cent. were adolescents (ages from thirteen to twenty-two). The largest number of those cases of adolescent insanity were cases of dementia præcox. From a total of 148, 120 patients are grouped under three of Kraepelin's varieties of dementia præcox, as follows:—dementia simplex, 11; hebephrenia 18; and katatonia, 91. An interesting analysis of all the cases of adolescent insanity is included in the paper. No comparison of the several forms of insanity is made with those found in European countries to make the paper fulfil the author's hope that its study will serve as a study of racial psychiatry.

HAMILTON C. MARR.

HOMICIDAL MELANCHOLICS. (*Homizide Melancholiker.*) EMIL
(392) RAIMANN (Vienna), *Arb. a. d. Neurol. Instit. a. d. Wien. Univ.*,
Bd. xvi., Teil 2, 1907.

THE writer of this article, while in attendance during two and a half years at the Provincial Criminal Court in Vienna, noticed the four cases which form the subject of his paper.

All the cases were afflicted with hypochondriacal melancholia, and showed homicidal tendencies. These tendencies were not blind impulses to attack surrounding objects, but were carefully planned out and premeditated. Three of them ended in brutal murder, and in the fourth case the victim was severely injured. In each case suicide as well as homicide seems to have been planned by the patient beforehand, and the failure to commit suicide (although in two cases attempts were made) is commented on. It is remarkable that some of the patients made actual attempts to save their own lives after committing the murder. In every case there was a family history of insanity, and in two a personal history of alcoholism. The peculiarity of the murder in each case was that the guilty person looked on it as an altruistic deed, intended to spare the loved one some harm.

HAMILTON C. MARR.

EYE FINDINGS IN GENERAL PARALYTICS. (*Augenbefunde bei (393) Paralytikern.*) H. DAVIDS, *Monatschr. f. Psychiat. u. Neur.*, Bd. xxiii., Ergänzungsheft, S. 1.

THIS article gives the results of a research undertaken to check the recent conclusions of Raviart, Pansier, de Montiyel, etc. As, however, it concerns only 26 patients—not a tenth of the number of the other observers—the results are of only limited value. The light reflex was affected in 92·3 per cent. of the cases, the accommodation reflex in 57·7 per cent. Changes in the fundus—especially the cloudy retina described by Uhthoff—were found in only 23 per cent.

ERNEST JONES.

KORSAKOW'S DISEASE. (*Die Korsakowsche Krankheit.*) W. (394) SERBSKY (Moscow), *Arch. a. d. Neurol. Instit. a. d. Wien. Univ.*, Bd. xvi., Teil 1, 1907.

PROFESSOR SERBSKY is of opinion that the discovery of Korsakow's disease is the greatest conquest that has been made of late years in the world of Psychiatry, and in importance can only be ranked with general paralysis. Both illnesses are well defined, and are based on exact anatomical and pathological data, although, unlike general paralysis, Korsakow's disease is rare. The paper is a historical analysis of the disease. The name of Korsakow was first associated with it by Professor Jolly at the International Medical Congress in Moscow (1897). The mental symptoms now grouped under the heading of Korsakow's disease have long been noticed in connection with all forms of multiple neuritis, no matter what their origin. To dispel a generally accepted opinion that the polyneuritic psychoses associated with septic processes are not due to septicæmia, but to alcohol, which was largely prescribed by medical men in such psychoses, a detailed summary of "A Case of Polyneuritic Psychoses with Autopsy" is given. The case in question formed the subject of a paper by Korsakow and Serbsky. Details of cases of multiple neuritis other than that of alcoholic origin are also included in the paper, and a summary by Dupré of the numerous causes of the disease. The latter summary is of a similar kind, but not so well arranged as Gower's classification of the causes of multiple neuritis.

One feature not constant in Korsakow's disease is referred to, viz., retrograde amnesia. This, according to the author, is only found in cases where, at the beginning of the illness, confusion of mind and fantastic pseudo-reminiscences appear. In such cases a deep-lying disturbance of the cortex is surmised.

In differentiating Korsakow's disease from other illnesses with like symptoms of amnesia and pseudo-reminiscences, besides the neuritic symptoms, an important characteristic of the disease may be observed, viz., the retention of the patient's character and personality (except during the period of temporary confusion). The patient retains his former sympathies and antipathies and is able to judge and conduct his affairs properly—all circumstances being otherwise favourable, and material at hand to aid the memory.

Korsakow's disease does not represent an amnesia, but amnesia in combination with neuritis and the retention of the patient's personality.
HAMILTON C. MARR.

SYPHILOMANIA AND SYPHILOPHOBIA. (*De la syphilomanie et (395) de la syphilophobie.*) C. AUDRY, *Ann. de dermat. et de syph.*, March 1908, p. 129.

AFTER a brief review of the literature of this interesting subject, which has received but scant attention from neurologists or alienists, the author defines the terms syphilomania and syphilophobia, and discusses their ætiology, symptomatology, prognosis, and treatment. Syphilomania is the obsession of the idea of syphilis in non-syphilitic subjects, or in those who have no serious reason to believe that they are infected. In syphilomania the obsession is permanent, irresistible, and incurable. It differs from syphilophobia in that the idea of syphilis is confused and vague. Rarely if ever neurasthenic, the subjects of syphilomania are sometimes psychasthenic, but as a rule they are degenerates of the ordinary type or victims of hysteria. Grave forms occur in which the obsession is sufficiently imperious to determine dangerous acts of insanity. Suicide, however, is rare, contrary to what occurs in syphilophobia.

Syphilophobia is the obsession of the idea of syphilis in syphilitic subjects, or in non-syphilitic subjects who have just grounds for believing that they are infected. Except in the grave forms the obsession is not continuous. It may last or recur during a period varying from six months to ten years or more, but the patient enjoys periods of relative repose, during which the latent obsession loses its power. The discontinuity of the fixed idea is an important diagnostic feature which allows one to distinguish ordinary syphilophobia from syphilomania, or from acute grave syphilophobia.

Syphilophobes may be normal though impressionable individuals or sufferers from psychasthenia or neurasthenia, but they are never, or very rarely, degenerates. Unlike syphilomania, syphilophobia is most frequent in males. The general culture and social position is higher than in syphilomania. Syphilitic infection and

its evolution in a predisposed soil are not sufficient to determine syphilophobia. A third factor is required, and is furnished by the suggestion relating to the dangers of syphilis brought to bear upon them, not only in quack advertisements, but also in the lay press and on the stage. All syphilophobes can be made to recognise that they are victims of a mental disorder against which they declare themselves powerless to react. This consciousness of their condition is never found in syphilomania. In syphilophobia the fixed idea is manifested in various forms, but is always related to a precise though not exact notion of syphilis. Some dread the external manifestations, some the nervous complications, others are afraid of spreading the disease, or imagine they can find traces of it in their wives or children. As a rule there is no relation between the intensity of the syphilitic infection and the syphilophobic reaction. A mild attack of syphilis may produce violent syphilophobia.

The grave form of syphilophobia is one of the most redoubtable complications of syphilis. Fortunately it is very rare. Audry found it present in only 5 out of 5000 patients. As a rule it is a late symptom, so that the question arises as to whether commencing arterio-sclerosis does not play an active part in its production.

In the treatment of syphilophobia the daily life of the patient should be carefully regulated, so that he should not be left a moment unoccupied. Audry has sometimes obtained good results by a substitution cure, *i.e.* by prescribing some absorbing occupation to take the place of the fixed idea. J. D. ROLLESTON.

MENTAL DISEASES AMONG JEWS. (Über die Geistesstörungen (396) bei den Juden.) MAX SICHEL, *Neurol. Centralbl.*, April 16 1908.

To the *Neurologisches Centralblatt* of April 16th, Dr Max Sichel, Assistant Medical Officer of the Frankfurt State Asylum, an institution which, for several reasons, offers a favourable field for such inquiries, contributes an interesting paper on the relative frequency of insanity among Jews. It has been maintained by many writers, *e.g.* Kraepelin, Mendel, Krafft-Ebing, Lombroso and others, that the Jewish race suffers to a disproportionate extent from mental maladies. Dr Sichel's inquiries lead him to the opposite view. Taking the statistics of his asylum from 1897 to 1907, he finds that, contrary to the general view, the proportion of Jewish insane to Jewish population is rather less than the non-Jewish proportion, but that wide differences exist in the proportions obtaining amongst the several clinical forms. In the Frankfurt Asylum in 1906 and 1907 the Jewish admissions

formed 6·5 per cent. of the whole, corresponding closely to the proportion of Jews (6·8 per cent.) in the general population of Frankfurt. If, however, from the total (Jewish and non-Jewish) admissions all cases with alcoholic psychoses be deducted, the proportion of Jewish admissions rises to 9·2 per cent. of the whole admissions, non-Jewish alcoholic admissions forming 32·0 per cent. of the non-Jewish, and the Jewish alcoholics only 1·5 per cent. of the Jewish admissions, and this notwithstanding that the Jews, Dr Sichel says, are large consumers of alcohol.

In every other clinical category—with the exception of epileptic psychoses, in which the Jewish proportion is one-half that of the non-Jewish—the Jews are relatively in excess, dementia præcox being 28·1 per cent. as compared with 16·6 per cent. for the non-Jewish; manic-depressive insanity, 11·7 per cent. as compared with 2·7 per cent.; general paralysis, 12·5 per cent. as compared with 8·3 per cent.; and hysteria, 10·9 per cent. as compared with 6·1 per cent. for the non-Jewish.

Pilcz, in his paper on "Mental Diseases in Jews," in which he bases his discussion on the statistics furnished in Vienna, expresses his doubts as to the value of any comparative statistical study which leaves out of account the figures of those in private asylums or in family care, and Dr Sichel's facts, though interesting, are open to the same objection.

R. CUNYNGHAM BROWN.

Reviews

NEW CLASSIFICATION OF CRIMINALS. (*Nuova classificazione dei Delinquenti.*) JOSÉ INGEGNIEROS (of Buenos Aires), *Biblio. di Sci. soc. e polit.*, N. 65. Remo Sandron, Milan, Palermo, and Naples, 1907, pp. 80.

PROF. INGEGNIEROS criticises the fundamental conceptions of what he calls the classical school of penal justice, according to which crime is regarded as the anti-juridical conduct of individuals in whom free-will and responsibility are assumed, and to whom punishment is meted out according to the gravity of their offence against abstract ideals, with little consideration of the environment and psychophysical constitution of the individual criminals. The author—who might be described as a deterministic "whole-hogger," inasmuch as he denies free-will to the sane as to the insane or to the moral imbecile—maintains that the ends of justice are defeated and the issues obscured by such metaphysical conceptions,

and would substitute for the ordinary penal sentences a scale of reformatory, repressive, and eliminative measures more in harmony with modern criminology.

He treats of criminology under the headings of (1) the ætiology of crime, that is the study of the determining factors of crime; (2) clinical criminology, *i.e.* the morphology, psychology, and degree of inadaptability to social environment of the criminal; and (3) the therapy of crime. Under this last head, in the treatment of which subject he makes the somewhat sweeping statement that a strict determinism is the necessary outcome of biological science, he of necessity does away with punishment in the sense of retribution altogether, and substitutes instead three degrees of maximum, medium, and minimum penal repression. Under maximum repression are included the "born" criminal, the insane criminal, the habitual and incorrigible criminal, the insane criminal with psychoses which have acquired permanent form, and alcoholic, epileptic, impulsive and incurable criminals. Under medium repression are grouped the corrigible but habitual criminal, the insane delinquent with curable psychoses, and the impulsive criminal with educable inhibition. Under minimum repression are included the occasional criminal, delinquents with transitory mental disorders, and criminals with "accidental disboulia." From the foregoing it is obvious that the author regards the criminal act only in so far as it affords an indication of the psycho-physical constitution of the individual actor or throws a light on his previous training and environment, and that whilst the epileptic convicted of petty larceny and the insane murderer would be classed together for maximum repression, the murderers "*per disboulie accidentali*" (a fairly large class) would be subjected to only the lowest scale of repression. This small book is not a study, but a by no means closely reasoned discourse, which, notwithstanding its title, contains no new idea.

R. CUNYNGHAM BROWN.

THE INFLUENCE OF ALCOHOL AND OTHER DRUGS ON FATIGUE. By W. H. R. RIVERS, M.D., F.R.C.P. London: Edward Arnold, 1908. Pp. 136. Price 6s. net.

THIS volume consists of the Croonian Lectures delivered at the Royal College of Physicians in 1906, with some change of arrangement and the addition of some new matter derived from later work. The drugs chiefly studied are caffeine and alcohol. Critical accounts are given of the work done with these by previous writers, and the author's own investigations are clearly described. The ergograph is used to show the course of muscular fatigue, while mental fatigue is estimated by multiplication, typewriting,

or MacDougall's new method of marking dots passed over a rotating cylinder and seen through a slit. The history of the meagre researches extant on the effects of strychnine, cocaine, tobacco, and other drugs is given, and some fresh experiments with these are described.

The special value of the new experimental work brought forward in this volume is found in the care taken to separate the specific effect of the drug from effects due to certain other factors, such as interest and sensory stimulation. With this end in view the drug was administered in such a form that it could not be distinguished from control mixtures which were given on the days when the drug was not taken. The subject of the experiment thus always took some draught, but did not know till after the records were completed whether the special drug studied was contained in it or not. As a result of this precaution the records of Dr Rivers show much less marked variations consequent on the ingestion of the drug than do those of most previous investigators. With alcohol, for example, except with large doses, the results are purely negative, whereas former experimenters had always found even small quantities followed by an immediate stimulating or depressing effect on the muscles.

By the emphasis which he lays on the necessity for control mixtures—a necessity which has been strangely ignored by most other workers—Dr Rivers has taken a noteworthy step towards the simplification of the problem before us. Apart from this question of method, his careful résumé of the results already attained, his description of his own experimental work and the conclusions he deduces therefrom, are of the utmost value both as marking the level of knowledge already attained, and as indicating the lines on which fresh research should proceed.

MARGARET DRUMMOND.

NUCLEI OF THE HUMAN SPINAL CORD. (Über die Kerne des menschlichen Rückenmarks.) L. JACOBSON, *Aus dem Anhang zu den Abhandl. d. Königl. Preuss. Akad. d. Wissenschaft, vom Jahre 1908*, pp. 72, plates 9.

FROM an examination of serial sections of an adult spinal cord the author classifies the various cells of the cord into the following groups and sub-groups. Plates showing the characteristic arrangement for each segment accompany the article.

I. Motor nuclei of the anterior cornua.

- (1) A medial group, the whole length of the cord down to S4, sometimes divisible into antero-median and postero-median. Stray cells in the anterior commissure.

(2) A lateral group. C1-D2 and L1-S3.

In the upper region—

(a) Antero-lateral.

(b) Internal intermedio-lateral or central and external intermedio-lateral.

(c) Internal and external postero-lateral.

In the lower region—

(a) Antero-lateral.

(b) Postero-lateral.

(c) Intero-lateral or central.

(d) Anterior (in L3 and L4).

(e) Retrodorsal (near reticular angle).

Cells of the lateral group occasionally occur in the white matter.

II. Sympathetic nuclei.

(1) Superior or thoracic lateral sympathetic nucleus, lower C8 to upper L3. Segmented arrangement, 2 parts—apical in the lateral horn or corresponding region; pre-angular in the angle between anterior and posterior horns.

(2) Inferior or sacral lateral sympathetic nucleus, lower S2 to coccygeal. Segmented like the upper, but the cell-nests are shorter. An external wedge-shaped group, and sometimes an inner group.

(3) Inferior or lumbo-sacral medial sympathetic nucleus, L4 to coccygeal. In lowest sacral borders with (2). At first confined to median border of anterior horn, in S2 extends to anterior border of the horn, in lower S3 and S4 occupies whole anterior horn and central area.

All three groups show (a) cells in small compact groups; (b) cells small, rounded, club-shaped or polygonal; (c) homogeneous appearance and fairly darkly stained.

III. Large-cell nuclei of the posterior horn.

(1) Basal or spino-cerebellar (Clarke's column), C8-L3. Occasional cells both above and below. A continuous column.

(2) Central. In centre of the posterior horn between its base and the substantia gelatinosa and in the so-called cervix and caput. Some cells in the cervical cord, few in the dorsal, in lumbo-sacral more abundant—almost a regular nucleus. Course of cell-fibres unknown.

(3) Pericornual.

(a) Apical. Whole cord except lower sacral; a few cells on margin of the tip of the posterior horn.

(b) Reticular. On the outer margin of the horn, from the tip to the reticular angle, S2-S4 especially.

IV. "Nucleus sensibilis proprius."

Gierke's cells. The small cells of the substantia gelatinosa. Abundant and constant throughout the whole length of the cord.

V. "Tractus cellularum."

Small cells scattered irregularly over the whole of the grey matter. The term "tract" is used because no sharply-defined groups or nuclei can be distinguished, but they may be conveniently divided into the following three series:—

- (a) Antero-median. Along the inner border of the anterior horn; may extend into the centre of the horn or into the central area at the junction of anterior and posterior horns.
- (b) Postero-median. The smallest both in number and size of cells. Situated in the region of Clarke's column, either actually among its cells or on any side of it. May also extend towards the central area of the grey matter.
- (c) Lateral intercornual. The largest numerically. Cells in the outer part of the posterior horn (anterior to the substantia gelatinosa). In the region of formatio reticularis and angle between anterior and posterior horns, and extending from these into the central area of the grey matter.

J. H. HARVEY PIRIE.

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Review of Neurology and Psychiatry

Original Articles

ANEURISM OF THE ANTERIOR CEREBRAL ARTERY, WITH UNUSUAL PROLONGATION OF LIFE AFTER RUPTURE: AUTOPSY.

(With Plates 22 and 23.)

By ALEXANDER BRUCE, M.D., F.R.C.P.E., Physician to the Royal Infirmary, Edinburgh; J. H. HARVEY PIRIE, B.Sc., M.D., Clinical Tutor in the Royal Infirmary, Edinburgh; and W. KELMAN MACDONALD, M.B., Ch.B., Resident Physician in the Royal Infirmary, Edinburgh.

THE following case of rupture of an intracranial aneurism appears to us to be of sufficient interest from the clinical and pathological aspects to merit publication :—

A. R., single, 33 years of age, smith, was admitted to the Royal Infirmary on 18th April 1908 in a state of unconsciousness, which was said to have lasted about two hours. At 11.30 A.M. he had become suddenly ill, and the foreman, who had been called to see him, found him at his bench leaning forwards and supporting himself by his extended arms. His face was flushed, and beads of perspiration stood on his forehead. He had been ill for three or four minutes, was quite conscious, and said that he had had a similar attack one week before from which he had recovered in a few minutes, as he expected he would be able to do in this case. He complained of great pain in the head and back of the neck, and of great giddiness. With assistance he was able to walk out into the open air. He sat down for about ten minutes, and, feeling better, started to walk home in company with a boy. After he had gone about 300

yards, however, he was unable to proceed further, and the foreman, on being called by the boy, found him lying unconscious in the street, assisted him into a cab, and brought him immediately to the Royal Infirmary, which he reached in a state of complete unconsciousness.

When examined on admission, patient was lying on his back with his arms by his sides and his legs extended. The face was slightly congested, the eyelids half closed, and the palpebral apertures equal in size. The pupils were contracted, quite circular, and equal in size. They reacted to light only to a slight degree, if at all. Both conjunctivæ were quite insensitive to touch. The eyeballs were not prominent, and did not move at all. The mouth was shut. There was a small amount of frothy saliva between the lips. The lips were not livid, but were blown out with each expiration. The breathing was irregular both in rhythm and in depth of inspiration, but there was no stertor. Coarse rhonchi in the lungs masked the character of the breathing, but it was possible on auscultation to hear a slight systolic murmur at the mitral area. There was no abnormal percussion dulness, and no want of expansion of the chest, and, as far as could be ascertained from sounds made on respiration, no alteration of vocal fremitus or resonance.

The arms lay along the sides of the trunk, but there were frequent spasms in which they were forcibly extended at the elbow and drawn to the side of the chest, or even in front of it. The forearms were very forcibly pronated, so that the backs of the hands were directed towards each other. The wrists were flexed, and the fingers were fully flexed, causing the hand to be tightly clenched, the thumbs being strongly adducted against the index fingers.

During these tonic spasms the lower extremities were tonically extended, and there was slight extension at the ankle joints and inversion of the feet. The lower extremities were not affected during every seizure.

The spasms lasted a few seconds, were usually accompanied by a loud expiratory effort and puffing out of the cheeks, but the facial muscles were otherwise not involved.

The breathing was at the rate of about 30 per minute, and was quiet in the intervals between the spasms. The mouth was kept shut, and any attempt to open it was strongly resisted.

ANEURISM OF ANTERIOR CEREBRAL ARTERY 451

The head tended to rotate to the left side, and the chin was approximated to the left shoulder. The attitude of the patient after each spasm was almost exactly that represented by Pl. 22, Fig. 1.¹ The position of the arms during the spasm is very similar to that depicted in Fig. 175 in Vol. II. of Gower's "Diseases of the Nervous System," 2nd Edition, which represents the phase of tonic immobility or tetanism during the first or epileptoid period of hysteria major, and is reproduced from Richer's "Études Cliniques sur l'Hystero-Epilepsie ou Grand Hysterie." The shoulders were less raised and the contractions less intense, but the relative positions of the forearm and hands, and the clenched attitude of the fingers, were similar in both cases.

The reflexes in the lower extremities showed a slight exaggeration. There was ankle clonus on both sides, but both plantar reflexes were of the flexor type.

The axes of the two eyes were not parallel, there being a slight divergent strabismus.

The pulse was about 70 per minute, regular in rhythm, of good force and moderate expansion. The tension, as measured by Gaertner's tonometer, was 95 mm. Hg.

The temperature was 96·4.

It was ascertained from his relatives that, with the exception of the attack above referred to, his previous health had generally been good, but that there was a slight tendency to tuberculosis in his family history.

The condition remained more or less as above described from about half-past twelve until three o'clock. The respirations tended to assume the Cheyne-Stokes character. The temperature remained about 96·6; the patient still lay on his back, breathing noisily and rapidly. By 3.30 the pulse had risen to 100; the respirations were more definitely of a Cheyne-Stokes character, and at the height of the cycle the arms and legs tended to become rigid and the back to be arched. At that time the temperature, which had begun to rise, had reached 98·2°, and by four o'clock had registered 100·8°. There was free

¹ This is not a photograph of the patient himself, but is one of the case observed by one of us (A. B.), and described by Bruce and Drummond in the *Review of Neurol. and Psychiat.*, vol. ii., p. 737. It had been mislaid when that case was published.

perspiration on the face and over the front of the chest. The patient began to perform lateral and vertical chewing movements.

By 4.30 P.M. the temperature had reached 101.4° ; the pulse was 110; respirations 45 per minute on an average, varying from 30 to 65 per minute according to the phase of the cycle at which it was counted. The Cheyne-Stokes character was then even more pronounced.

As the temperature was rising so rapidly and ominously, ice was applied to the trunk and limbs, with the result that the temperature fell from 101.6° to 97.6° , and the pulse from 120 to 90, the respirations remaining at 48 per minute. After the application of the ice, the spasms and chewing movements ceased. Loud rhonchi were heard over all the chest, obscuring the heart sounds.

The urine, which had been drawn off by catheter previous to the application of the ice, had a specific gravity of 1033, and showed a trace of albumen, but no other abnormal constituent.

Lumbar puncture was performed, and 25 c.c. of a brownish red fluid were drawn off under high pressure. This fluid was obviously composed of blood and cerebro-spinal fluid mixed, and, after standing in the test-tube, the solid elements began immediately to settle, and the precipitation was almost completed in two hours. On examination it was found that albumen was present in large amount. No sugar was present, but there was a distant though partial reduction with Fehling's reagent, a greenish colour with flaky precipitate being produced on heating. Blood was present, the guaiac test being positive. On microscopical examination the films showed:

(1) Large number of crenated red blood corpuscles; (2) a few polymorpho-nuclear leucocytes; (3) a few lymphocytes; and (4) a few large mononuclear cells.

The red blood corpuscles were the most striking corpuscular elements in the films. Many were perfect in shape and size, and many were reduced to the merest broken-down shell of an erythrocyte. All gradations between the two limits were found, very many of the corpuscles being beautifully crenated, although the fluid was microscopically examined as soon as it was obtained. The white cells present were polymorphs, lymphocytes, and large mononuclears: none of those which were phagocytic showed any organ-

isms in their interior. In some films there were some staphylococci (few) and a few rod-shaped organisms, but none were very definite, and none were intracellular. Their presence was probably due to accidental contamination. When the fluid was cultured, these organisms proved to be staphylococci (albus and aureus).

At 8.30 the temperature was still 98.8. The patient was perspiring freely. The pupils were moderately dilated, the right being greater than the left. Their outline was not quite circular. The jaws were firmly clenched.

At 9 P.M., the breathing was much quieter, and the spasms had ceased since eight o'clock, with the exception of the chewing movements and some movements of the tongue. The conjunctival reflexes had returned on both sides.

By 9.20 the pupils had begun to react to light, and the breathing had become more quiet and regular, altogether without stertor. This improvement in the condition remained fairly constant until eight o'clock on the following evening, the 19th, when the patient looked considerably improved, but about 9.30 P.M. the face became more congested and cyanosed, the perspiration stood in beads on his forehead, the temperature suddenly fell from 98° to 96.8°, and by 10 P.M. it had fallen as low as 95°. During the same time the respirations had increased from 38 to 48 per minute, and the pulse from 124 to 160, and at times it became so frequent that it was difficult to count it. The arms lay flaccid by the sides of the trunk, free from spasms or twitchings of any kind. The pupils were dilated, but were circular and equal. The contraction to light was practically gone, and the conjunctivæ were quite insensitive. The coma was now much deeper than on admission.

Hot water bottles were placed round the patient, and the pulse gradually improved in strength and slowed down to about 150. The respirations became deeper and less noisy than they had been, and the temperature began again to rise, becoming normal by 11 P.M. By midnight the immediate danger seemed to have passed, but the coma continued. Rhonchi, sonorous and sibilant, were heard all over the chest, and slight percussion dullness appeared at the bases posteriorly. Patient was fed with egg and milk by means of the nasal tube.

By 4 A.M. of the 20th, the patient had further improved. His temperature was 97.6°; respirations, 32; pulse, 130. The

pupils both reacted slightly to light, and were circular. The conjunctivæ were again sensitive.

A certain degree of improvement in the condition continued until 4.30 P.M., when there was a slight aggravation of the symptoms. By 8 P.M. it was noted that the lower extremities were now quite flaccid, and no deep reflexes could be obtained. The plantar reflex was difficult to elicit, but was of the flexor type on both sides. The upper extremities were quite free from spasms. The condition of the heart and lungs remained as before.

On the 21st it was noticed that the flaccidity of the lower extremities was less marked, and that while there were no actual spasms in the upper limbs, the tonicity of the muscles was slightly increased.

At 6 A.M. there was a rise of temperature to 101, which was met by the external application of ice and iced water. During the forenoon the comatose condition gradually became less deep, the eyes were opened, the eyeballs moved, and an impression was made upon his relatives during their visits that he seemed to recognise them. The upper and lower extremities showed some degree of tonicity, and there was a slight, feeble response to stimulation of any part. The deep reflexes again reappeared, and were somewhat exaggerated. The superficial reflexes were all present, the plantar reflex being still of the flexor type.

The improvement which had set in during this day was maintained. The patient appeared to be conscious. Although he was not able to speak, he followed with his eyes the movements of his relatives who came to see him. It was thought that he made a voluntary attempt to expectorate the mucus accumulating in his pharynx.

On the 22nd the plantar reflexes became extensor in character; the rhonchi disappeared from the lung, and the percussion dulness from the base. The systolic murmur could no longer be recognised in the mitral area. The urine passed by the patient retained practically the same character as before.

From the 23rd to the 28th of April the patient lay in the condition above described. There were no convulsions and no spasms of any kind. The temperature varied somewhat, and each time it rose above 101° F. towels with iced water were applied. They invariably reduced the temperature to about 99°. On the 23rd of April iced towels were used 9 times; on the 24th,

9 times; on the 25th, 26th, and 27th, 7 times; and on the 28th, 5 times, and in every instance the application was followed by a fall of temperature. On several occasions the patient so far recovered as to seemingly recognise his friends, but he never spoke, although he frequently appeared to be on the point of saying something. When anyone approached his bed, he looked at the visitor and quite consciously followed his or her movements round the bed, the movements of the eyeballs being well carried out. At other times he was less conscious, and lay on the bed taking absolutely no notice of anything or anybody.

By a second lumbar puncture, made on the 27th, 15 c.c. of fluid was obtained. It flowed freely and appeared essentially similar in character to that previously obtained, and microscopical and bacteriological examinations gave similar results.

Examination of the blood showed that the red blood corpuscles were between 4,300,000 and 4,500,000. Hæmoglobin remained steady at 80 per cent. White blood corpuscles were: on the 23rd, 14,000; on the 24th, 17,000; on the 26th, 19,000; on the 28th, 27,000. The differential count showed about: polymorphs, 70 per cent.; lymphocytes, 19 per cent.; mononuclears, 10 per cent.; eosinophiles, 1 per cent. The average blood pressure by Gaertner's tonometer remained at 95°.

On the 29th the condition became more grave; the limbs became quite flaccid; the eyelids remained incompletely closed, the divergent strabismus being present, but the eyes tended to show asynchronous movements. The pupils were moderately dilated, the right being larger than the left. They did not react to light. The conjunctivæ were insensitive to touch. The upper and lower limbs were flaccid; there were no reflexes to pinching. Rhonchi and crepitations were again heard all over the chest. The coma deepened, and the patient died quietly at 6 P.M. on the 29th, having lived twelve days after the initial onset of the symptoms.

Autopsy.—The autopsy revealed evidence of incompetence of the mitral and tricuspid valves of the heart. There were on the cusps of the mitral valve several large nodules of old endocarditis on the opposed surfaces. The lungs indicated a degree of congestion in the lower lobes, and the kidneys showed evidence of slight sub-acute nephritis.

Spinal Cord.—The dura mater was somewhat tense. Under

the arachnoid a thin layer of blood-stained fluid covered the cord throughout its whole length. A few small calcareous plates were found in the arachnoid in the lumbo-sacral region. When the layer of blood under the arachnoid was removed, the meninges had a rusty-red tinge. There was great tortuosity of the veins of the posterior surface of the cord. The consistence of the cord was slightly reduced in the dorsal and cervical regions; in the lumbar region it was fairly firm. On section of the cord, in the lumbar region, the anterior cornua were found to be somewhat congested. There were no hæmorrhages visible to the naked eye. On dividing the upper extremity of the cord from the medulla, as soon as the arachnoid was incised a large quantity of blood welled out from underneath this membrane through the foramen magnum.

Brain.—On opening the skull, and dividing the dura mater, the remains of the blood which had escaped from the foramen magnum was found in the sub-arachnoid space, the sulci, especially on the right side, containing a large quantity of blood-stained fluid, and the summits of the convolutions in the lower frontal, temporal and opercular regions being separated from the arachnoid by a thin layer of blood. Over the whole of the vertex minute bubbles of gas were found in the sub-arachnoid space. The arteries at the base of the brain were all normal, and the veins of the vertex were greatly engorged.

On making a longitudinal transverse section through the brain below the level of the corpus callosum, it was found that the two septa lucida, the fornix and almost the whole length of the corpus callosum had been ploughed through by a hæmorrhage which had evidently arisen from between the lower part of the two frontal lobes and had passed upwards and backwards, its progress above being arrested merely by the pia arachnoid covering the corpus callosum between the two hemispheres. Some of the blood had also entered the lateral ventricles, and had coagulated there without, however, either tearing up or even seriously compressing the basal ganglia. The blood did not appear to have passed backwards through the aqueduct of Sylvius into the fourth ventricle. The main mass of the extravasated blood had evidently found its way directly into the sub-arachnoid space, and had spread freely through this space along the lines of least resistance, filling up the sulci, the sub-

arachnoid cisterns, and raising the membrane from the convolutions, passing backwards, and, to some extent, seeking an entrance into the fourth ventricle from the foramen of Majendie, and also passing along the spinal cord under the arachnoid.

On making transverse sections through the basal half of the cerebrum, after the brain had been carefully hardened in a 7 per cent. solution of formalin, a firm clot of blood was found between the two frontal lobes. This had evidently been the starting-point of the great extravasation just described, for when a part of the clot was carefully washed away by a gentle stream of water, an aneurism on the left anterior cerebral artery was exposed (Pl. 22, Fig. 2). This aneurism was almost spherical in shape, and about the size of a small pea—5-6 mm. in diameter. It was carefully removed along with an adjacent portion of the brain cortex, and after some further fixation it was embedded in paraffin and cut into serial microscopical sections. From these it was ascertained that the aneurism was situated in the fork produced by the Y-shaped division of the anterior cerebral artery into two branches. It had formed on one of these branches just at the point of division (Pl. 23, Fig. 3). Beyond the limits of the aneurism, the main artery and its branches were almost completely normal in their structure, except at two points, which will be immediately referred to. There was no trace of any thickening or degeneration of any of the coats of the arteries, and, as already stated, the arteries at the base of the brain were almost normal.

The aneurism was seen to be composed of two parts: (1) a small primary sac (*b c*, Pl. 23, Figs. 3, 4, 5) formed by a local dilatation of the wall of the artery from which it arose; and (2) a somewhat larger secondary sac, or false aneurism (*d* in Figs. 4, 5, and 6), which had arisen by rupture from the preceding sac. Its walls were formed merely by the condensed tissue of the pia arachnoid, and its cavity was almost completely filled by mixed blood clot. On one side of this there was a further coagulum formed by blood which had escaped into the general mesh-work of the pia arachnoid (Figs. 5 and 6).

Microscopical examination of the wall of the aneurism indicated that the *initial weakening of the vessel which led to its formation was due to a localised defect in the muscular coat*. This was most clearly evident at a part of the wall at which

dilatation was just commencing, and where other changes had not been produced to complicate the picture, and the same condition was also observed in one or two small aneurismal dilatations of the other branch of the anterior cerebral artery at a little distance from the main division. The nature of the defect of the middle coat is shown in Fig. 3, and also in Figs. 7 and 8. At *c* in Fig. 3, and in Fig. 7, which is a more highly magnified view of the same part of the wall of the sac, it is seen that the middle coat is simply absent, and that the adventitia and the intima are in direct apposition. There is no indication of any pathological change in either the adventitia or the intima of such a nature as to have been capable of causing the disappearance of the media, and it is seen that at the margin of the sac the middle coat has become somewhat rapidly thinned or tapered off. Over the sac it is entirely absent until the opposite margin is reached, when it again begins to appear, and to increase in thickness in the same manner as at the opposite side. The adventitia in no way differs from the normal. The intima is slightly thickened by the proliferation of its endothelium, and by the splitting up of the elastic lamina into a series of layers which, on cross section, appear as tortuous fibrils (see Fig. 8).

Where the bulging of the sac has reached a higher degree, further changes in the intima have developed, which obscure the nature and meaning of the changes in the middle coat. The intima here (Fig. 3, *b*) has undergone an exceedingly high degree of thickening by proliferation of its endothelium. Its boundary from the remnants of the media, can be best determined in sections which have been stained for elastic tissue by Weigert's fuschin-resorcin method. It appears from these that at the two margins of the sac (*b*), the elastic lamina, split and tortuous, as already described, comes for a short distance into immediate contact with the adventitia, and then dips down between the thickened intima and the remaining laminae of the muscular fibres of the middle coat. Even here the remaining portion of the middle coat is distinctly defective.

The wall of the more fully formed part of the primary sac showed a great and irregular thickening of the endothelium of the inner coat, with here and there small remnants of the elastic lamina and slight thickening of the adventitia, but practically

no indication of the presence of non-striped muscular tissue. At one part of the wall of the sac a small rupture had taken place and the blood had escaped into the pia arachnoid tissue, which it had driven before it and condensed into a firm layer sufficient to resist, in the first instance at all events, the further progress of the extravasated blood, which had to a large extent become coagulated within its sac. A small channel had, however, been left through which the blood had escaped into the loose-meshed sub-arachnoid tissue. A small part of this had coagulated, and is shown at *e*, Figs. 4 and 5.

It was found that on the branch α' of the anterior cerebral artery there were one or two points at which similar defects of the middle coat were associated with slight aneurismal dilations. One of these is seen at *i*, Fig. 6. This, as in the previous case, was situated near a point of division of α' .

With regard to the ætiology of the condition, previous observers, such as Oppenheim (2), Beadles (3), etc., have drawn attention to the frequency with which aneurisms of the larger cerebral arteries arise from their points of division and from certain other sites of predilection, and to the relative frequency of the occurrence of such aneurisms in young individuals in whom there is no other evidence of arterial disease. The fact that in this case there was complete absence of anything to indicate a reason for the local disappearance of the middle coat, seems entirely to favour the view that the predisposing cause of the aneurism was a congenital defect of this coat.

From the clinical aspect, the case was of unusual interest in several respects: (1) The occurrence of a transitory attack of giddiness, with headache a week previous to the commencement of the fatal illness; (2) the sudden onset with unconsciousness associated with convulsive seizures of a special character without, in the first instance, any definite paralysis on either side of the body, but with a later temporary flaccidity of the muscles; (3) the large amount of blood-stained cerebro-spinal fluid obtained by lumbar puncture on two occasions; (4) the duration of life for twelve days after the first onset; and (5) the apparent partial recovery during the illness.

With regard to the first transitory attack of giddiness, it appears not improbable that this may have been associated with either the first development of the secondary sac or with a slight

rupture of this sac and the escape of a limited quantity of blood into the pia arachnoid. If so the quantity that had escaped must have been comparatively small, for it did not impair the patient's faculties in any way.

The clinical history seems to indicate that there must have been at least three other occasions on which hæmorrhages had occurred, namely, on the date of admission, on the following day, and on the day preceding death, on all of which there was a marked aggravation of the symptoms, with signs of increased intracranial pressure.

The convulsive seizures so exactly simulated those observed by one of us (A. B.), and recorded in this *Review* (*loc. cit.*), as to suggest a similar cause. The patient's condition after the sudden onset seemed to indicate the probable existence of a hæmorrhage into the third and lateral ventricles, but a hæmorrhage which did not destroy the internal capsule on either side, as there was neither hemiplegia nor diplegia at first. In this case, therefore, it could not have been due to the rupture of any of the vessels in the basal ganglia.

The amount of blood removed by lumbar puncture was so much greater than that met with in ordinary cases of hæmorrhage into the ventricles as to suggest that the blood had descended into the spinal canal either under the dura or under the arachnoid. The presence of this large quantity of blood-stained fluid on lumbar puncture and the nature of the convulsions, as well as the initial absence of hemiplegia or diplegia, or of any of the symptoms of cerebral hæmorrhage, suggested during life the possibility of a rupture of an intercranial aneurism.

The duration of life after the onset was very remarkable, as in hæmorrhage into the ventricles such a duration is recorded only in a very small number of cases. Sanders (4), in his valuable work on ventricular hæmorrhage, has found that in about 65·7 per cent. of the recorded cases death has occurred within twenty-four hours, and in the great majority of these in less than twelve hours, rapid death being the rule and delayed death the exception. He refers to three cases in which death was deferred until the twelfth day, one in which it did not occur until the eighteenth day, and one in which life was prolonged for a month. In none of these cases was there any association with aneurism.

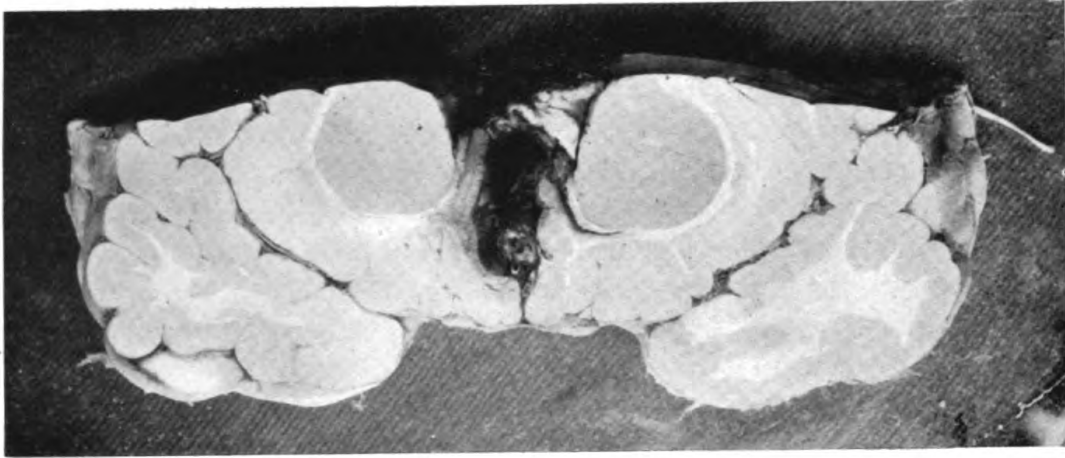


FIG. 2.



FIG. 1.

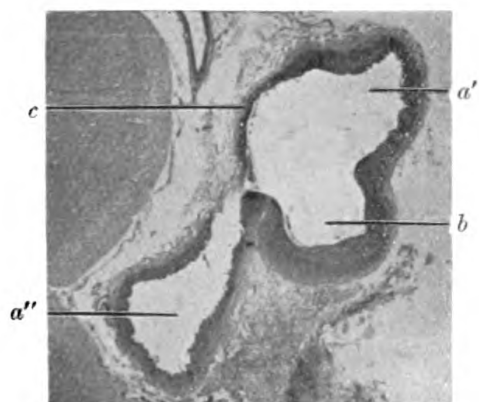


FIG. 3.

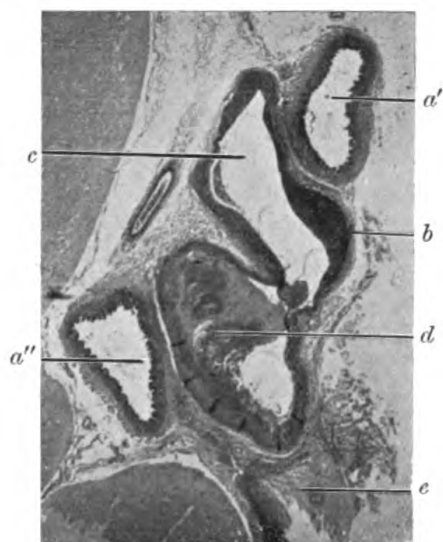


FIG. 4.

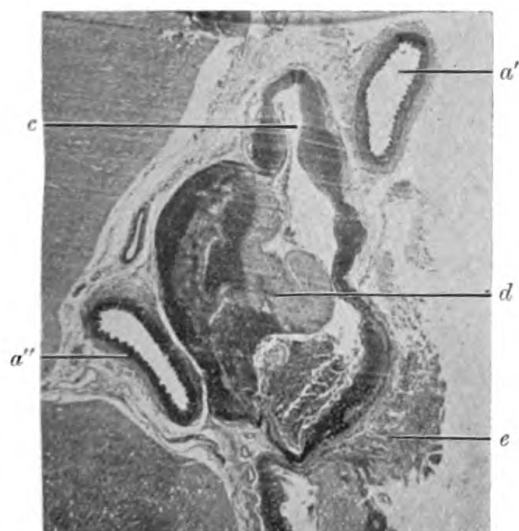


FIG. 5.

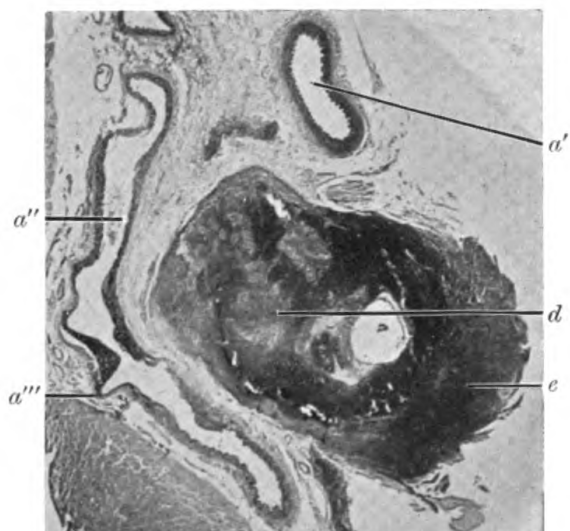


FIG. 6.

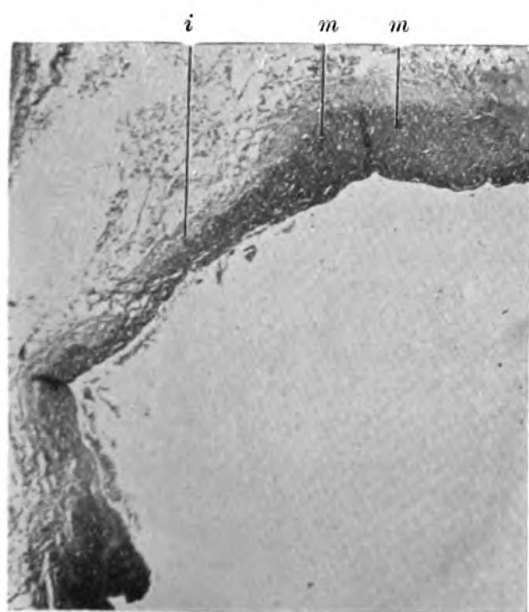


FIG. 7.

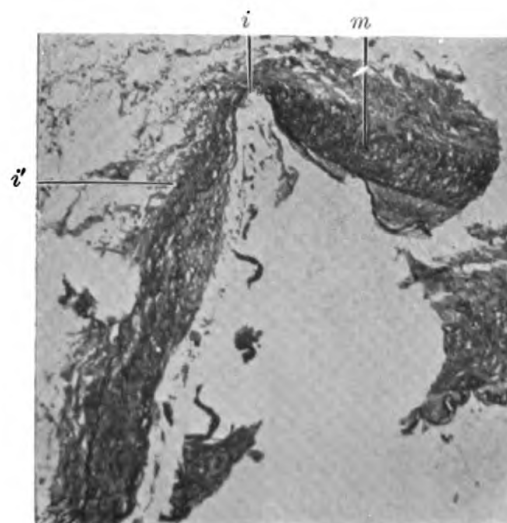


FIG. 8.

Sanders is inclined to explain the apparent improvement which appears to be by no means infrequent in cases of hæmorrhage into the ventricles, by the fact that the brain has become accustomed to the pressure, and that its function has thus become partially or completely restored. The improvement varies from a very slight degree to perfect restoration of consciousness. As a rule it is merely transitory and is followed by a relapse; it may be from a second hæmorrhage. In one case there was in all probability a succession of slighter or more severe hæmorrhages from the aneurism, to the pressure of some of which the brain had become partially accustomed by the displacement of cerebro-spinal fluid.

The microscopical investigation of this case was made in the Laboratory of the Royal College of Physicians.

DESCRIPTION OF FIGURES.

PLATE 22.

Fig. 1.—To show attitude of wrists and hands immediately after a fit. Photograph of case recorded by Bruce and Drummond (*Rev. Neurol. and Psychiat.*, Vol. ii., p. 737), and reproduced to show the similar attitude in present case.

Fig. 2.—Frontal section through anterior part of brain of A. R., showing the basal halves of both hemispheres separated by blood-clot (black), in the anterior part of which, and to the right side, is seen the aneurism as a paler globular mass.

PLATE 23.

a' and *a''*, branches of anterior cerebral artery; *b* and *c*, opposite sides of aneurism; *d*, secondary (so-called false) aneurism followed by rupture of the primary aneurism *b, c*; *e*, hæmorrhage into tissue of pia arachnoid from secondary sac; *i, i'*, intima of wall of vessel; *m*, media.

Fig. 3.—Shows the aneurism *b, c*, formed on one branch (*a'*) of the anterior cerebral artery at the point of division into two branches, *a'* and *a''*.

At *b*, the intima has undergone great thickening, while the media has become thinned.

At *c*, the middle coat has entirely disappeared (see Fig. 7).

Figs. 4 and 5.—Show the aneurism *b, c*, lying between the now completely separated branches *a', a''* of the anterior cerebral artery. By the rupture of the wall of *b, c* at its power part, a second sac has been formed. It is filled with partly-organised thrombus, and its walls are formed by condensed tissue of pia arachnoid.

At *e*, blood has escaped from the secondary sac.

Fig. 6.—*d*, The secondary aneurism, with thin walls, formed merely of condensed pia arachnoid tissue.

462 ANEURISM OF ANTERIOR CEREBRAL ARTERY

c, Hæmorrhage from the secondary sac into the pia arachnoid.

a' and *a''*, Branches of anterior cerebral artery.

a''', A small aneurism, with absence of middle coat.

Fig. 7.—(H. P. Zeiss Planar.) Part of wall of aneurism (*c* in Fig. 3), showing how the media, *m*, *m*, becomes rapidly thinned, and at *i* has completely disappeared, leaving merely the slightly thickened intima *i*.

Fig. 8.—(H. P. Zeiss Planar.) Section stained with Weigert's (resorcin fuchsin) stain for elastic tissue.

m, Media, terminated suddenly at the bend in the wall of the vessel at *i*, where the intima is alone present.

i', Intima thickened, and, when examined with hand lens, seen to contain numerous tortuous fibrillæ of elastic tissue.

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A CASE OF ARTERIO-VENOUS ANEURISM OF THE INTERNAL CAROTID ARTERY AND CAVERNOUS SINUS.

By ALEXANDER BRUCE, M.D., etc.

(With Plate 24.)

CASES of this condition in which a full clinical history is available do not appear to be very numerous, and therefore the notes of a case which has been under the writer's observation may be of interest.

The patient was a woman of 74 years of age. She had just recovered from an attack of influenza, complicated with pleurisy of ten weeks' duration, but previous to that time she had been a thoroughly healthy woman. One morning in the

end of May 1907 she was suddenly wakened about four o'clock by a loud cracking noise in her head. Much alarmed, she sprang out of bed and rushed into the next room, where she wakened her daughter. She was able to walk quite well, but complained of severe occipital headache and of noises inside her head like those of a steam-engine panting. Her daughter gave her some powder to allay the headache and to make her sleep, but without success; she was too excited to sleep any more that night. She remained in bed during the whole of the day after the attack and part of the following day, suffering from pains in the head and from the noises which she described as like the noise of a steam-engine. These noises never ceased except during her sleep since they first began to trouble her.

Shortly after the onset her walking became somewhat impaired. She lost power in her legs, which gave way under her when she tried to stand, and she could not move about without help.

About two weeks after the onset the eyes became painful, especially when she attempted to read. About a week later the right eye became congested and swollen, and a few days afterwards the left eye was similarly affected. Then in another month—that is about two months after the onset—the left eye gradually became more prominent, the conjunctivæ being very red. About the same time the right eyelid began to droop, and by the end of June the palpebral aperture was completely occluded. About the time of the beginning of the protusion of the left eye there seems to have been a certain amount of lividity of the face round about the eye.

Patient was admitted to Ward 40 in the Royal Infirmary, under the care of Dr George Mackay, on the recommendation of Dr Stewart. Ever since the onset she had been sleeping exceedingly badly, and shortly before admission she seems to have shown symptoms of a mild maniacal type.

After admission to Hospital the pain in the head became gradually worse, so that she could scarcely keep her head on the pillow because of the aching. The noises continued as before, the prominence of the eye-ball diminished slightly, but the ptosis persisted. The walking gradually improved, and the patient was able to walk without support. All the mental symptoms seemed to have disappeared, but the sleeplessness was

very persistent, owing to the continuance of the pain and the noises in the head.

The personal and family histories were perfectly satisfactory, and do not throw any light on the causation of the condition. They need not, therefore, be detailed here.

The patient was seen by me in consultation with Dr Mackay, and we discussed the question of the diagnosis, and of the possibility of treatment by ligature of the carotid artery. On further consultation with Mr Cotterill this plan had to be reluctantly abandoned owing to the patient's advanced age, and the extremely atheromatous state in which the carotid artery was found to be. The patient was subsequently placed under my immediate care by Dr Mackay, and the following notes are extracted from the ward journal.

The patient was spare and grey-haired, with slightly livid lips and complete left-sided ptosis. The left eye was enlarged and protruding (Pl. 24). There was congestion and œdema of the eyelids and conjunctiva. The pupil was moderate in size and the iris normal in appearance, but it did not react to light or to accommodation. The eye itself was absolutely immobile. The conjunctivæ were quite sensitive to touch. Vision was not materially impaired, the patient being able to see quite well when the eyelids were kept open. The veins of both retinae were congested, but there was no optic neuritis.

With regard to the right eye, there was nothing abnormal beyond the fact that the outward movement was somewhat impaired.

The patient complained of some degree of deafness, not of recent origin, in both ears. She suffered from constant and extreme pain all over the head, of a beating character, the beats corresponding to the noises heard in the head. The pain was generally worst at the back of the head, or on the part of the head which, for the time being, was most dependent. The pain was aggravated by external noises. There was tenderness to touch, especially over the supra-orbital and auricular nerves on the left side. The noises of which she complained were compared by her to the puffing of a steam engine. They were constantly present, but were aggravated by external noises, and by anyone speaking to her for any length of time.

On auscultation of the head a marked blowing musical

PLATE 24.



sound was heard more or less continuously, but with a strong reinforcement which was systolic in time. This murmur could be made out all over the vertex, sides and back of the head, but was most pronounced in the region of the temporal muscle (maximum at the pterion) *on both sides*—louder on the left side, if anything. This sound was conducted forwards with diminishing intensity to the external angular process of the orbit. It diminished also on going backwards, and could be heard only faintly above and behind the ears. It was conducted downwards along the line of the internal carotid artery, and was well heard right down the cheek as far as the lower jaw, with an increase in loudness over the malar bones. A pulsation was visible in front of and below the ears.

The patient's sleep was greatly disturbed by the noises and pain in the head.

Further examination of the nervous system did not reveal anything of importance. There was no paralysis or sensory disturbance. The deep reflexes in the lower extremities were not very active. They seemed to be more so in the upper extremities. The plantar reflexes were of the flexor type.

Examination of the circulatory system showed a slight degree of hypertrophy of the heart, with considerable hardening of the arteries, the carotids, as already mentioned, appearing like hard cords on each side of the neck.

While the patient remained in hospital she was treated with bromide and iodide of potassium, with rest in bed, and with the further addition of butyl chloral for the pain in the head. There was a considerable diminution of the pain, of the lividity of the left eyelid, of the chemosis of the conjunctiva, and of the protrusion of the eye-ball, but the murmur heard over the head and the noises in the ear continued unchanged up to the time of the patient's discharge from hospital on the 7th of December 1907.

It was subsequently learned that she died a few weeks afterwards, but unfortunately no opportunity was obtained for an examination.

In spite of the absence of an autopsy, there can be little doubt that the symptoms were due to the sudden development of a communication between the internal carotid artery and the cavernous sinus on the left side. Such communications may

arise either from the rupture of an aneurism or of a carotid artery whose walls have undergone a greater or less degree of atheromatous degeneration, but without dilatation, or they may be caused by an injury of the artery. It is almost impossible to account for the sudden crashing sensation in the head in this case, with the subsequent subjective noises and the auscultatory phenomena, in any other way than by a sudden rupture and subsequent communication of the two vessels. It may be presumed that the sudden noise in the head was coincident with the rupture into the cavernous sinus. Rivington, in his valuable paper on "Pulsating Tumour of the Left Orbit," in the *Medico-Chirurgical Society's Transactions*, vol. lviii., p. 184, shows that in the majority of the idiopathic cases there was a sudden onset with a crashing noise in the head, associated with sudden and severe headache. In two instances the patients were roused from sleep by the pain and noise. Rivington gives it as his opinion that a continuous murmur with associated systolic reinforcement, heard over the anterior part of the head, indicates the presence of an arterio-venous aneurism of the carotid and cavernous sinus. His statement (*loc. cit.*, p. 242) that a distinctly intermittent bruit would point to true aneurism must, in view of the work of Beadles, in *Brain*, 1907, be accepted with caution.

The murmur heard on auscultation was continuous, with a somewhat musical character and a definite systolic reinforcement—that is, it was distinctly louder during the systole. It was widely distributed, and it was interesting to note that the part of the temple at which it was loudest corresponded to the pterion, or to the great wing of the sphenoid. Dr Kelman Macdonald, who studied the distribution of the murmur with great care, suggested that the sphenoid was probably the most direct means of conducting the murmur to the surface.

Wildbrand and Saenger state that the protrusion of one or both eyes may be absent, or may be immediate, or may not appear for several days, weeks or months after the rupture. It was noteworthy that in this case it did not appear until three weeks after the onset, and that the congestion and swelling appeared on the right eye a week before the left eye was similarly affected.¹ The variation of the date of the appearance

¹ There was some reason—namely, the paresis of the right external rectus—to think that the right carotid artery might also have been somewhat dilated, but no evidence that it was in an aneurismal condition.

of this protrusion and congestion must depend upon several factors—the size of the orifice in the wall of the artery or of the aneurism, and the conditions regulating the possibility of the free escape of blood from the cavernous sinus in the proper direction. It will be obvious that should the opening in the arterial wall dilate beyond a certain limit, the various outlets of the cavernous sinus might thereby become insufficient to drain away the additional amount of blood forced into it, and thus an exophthalmos with chemosis and perhaps pulsation would be produced. On the other hand, it is possible that the size of the orifice might remain unaltered, but there might form in the cavernous sinus such a degree of thrombosis as to limit the free escape of blood from it, and in this way backward venous pressure upon the eye-ball, etc., with its various consequences, might be brought about.

It is to be noted that the fifth nerve alone of those that pass along the wall of the cavernous sinus escaped paralysis, the conjunctiva remaining perfectly sensitive. To this may be attributed the absence of any neuromyolytic keratitis, which appears to occur in a very high proportion (according to Wildbrand and Sanger in about 37 per cent.) of the cases.

A short note of some of the more important accessible literature is appended.

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Abstracts

ANATOMY.

"DER BAU DER SPINALGANGLIEN DES MENSCHEN UND DER (397) SÄUGETIERE." Dr A. S. DOGIEL, Professor of Histology in the University of St Petersburg. Jena, 1908, Gustav Fischer.

IN a monograph of 150 pages, accompanied by many beautiful coloured plates, Professor Dogiel discusses the structure of the mammalian spinal ganglion. The work is dedicated to "those distinguished investigators of the nervous system—Ramon y Cajal, Camillo Golgi, and Gustav Retzius," and an introduction to it gives a good review of the results which have accrued from the researches of these and other workers in this subject. The employment of the silver reduction method by Cajal confirmed many of the previous observations of Dogiel, and added new material to what was already known. Cajal found six distinct types of cells among the spinal ganglia, and these types are briefly criticised by Dogiel in the light of his own researches. The work of Levi, Lenhossék and Nageotte is reviewed, and particular notice is taken of the question of the regeneration of nerve fibres in the posterior nerve roots from cells of the spinal ganglia.

In his investigations Dogiel made use of Cajal's method in addition to that of staining living material by weak methylene blue, but found that he got better results by the latter method. A description is given of his technique, the difficulties of which would require much patience and practice to overcome. Hand-cut sections of the fresh ganglia are incubated at body temperature in weak methylene blue for a suitable time, fixed in 7 per cent. solution of ammonium molybdate for eighteen hours, then dehydrated, cleared and mounted in balsam or damar. The picture of the ganglion cells obtained by this method, as illustrated in the plates, is certainly very fine, and the process in the hands of a competent worker would seem to give a better general impression of the cell and its branches than can be obtained by the silver method. The latter is of more use in revealing the fibrillar nature of the cell-body itself.

Dogiel finds that the spinal ganglion is a much more complex structure than has previously been supposed. It contains at least eleven distinct types of cells, and between these types no direct relationship exists. The types are designated by Roman numerals, and in some of them there are sub-varieties. The cells are unipolar, bipolar and multipolar. To type I. belong the commonly described typical cells of the spinal ganglion. The cells of type

II. are characterised by the giving off of collaterals from the axis cylinder after it emerges from the connective tissue capsule of its cell, the collateral fibres ending in a form of end-plate which lies in the sheath of the cell or in the connective tissue of the ganglion. Many of the other types give off branches which end in different forms of swellings or end-plates in the ganglion. Type VI. contains four sub-varieties, and its cells are remarkable on account of the number of branches which arise from the cell; these constitute a thick network of fibres which branch and anastomose with one another, and finally unite to form an axis cylinder process which eventually divides into a peripheral and central branch. The cells of type VIII. are unipolar, but the peripheral branch of the axis-cylinder breaks up into numerous fibres which end in the neighbourhood of the ganglion or in the posterior nerve roots. Type IX. consists of bipolar cells which have preserved their embryonic character; the peripheral process is usually thicker than the one which runs centrally. Types X. and XI. are made up of true multipolar cells. In type X. the dendrites do not emerge from the connective tissue capsule of the cell. The cells of this group agree in many respects with the multipolar cell described by Cajal. Perhaps the most interesting are the cells of type XI., which have peculiarities distinguishing them from all nerve cells hitherto known. They are large or medium-sized, occurring in groups, or singly in the nerve trunk or connective tissue near the ganglion. One process is an axon which runs directly into the spinal cord without branching; the other processes become medullated fibres or remain non-medullated, branch after leaving the cell capsule, and end in the connective tissue framework of the ganglion or in some part of the posterior nerve roots. Dogiel believes that these processes represent the peripheral branch of the other ganglion cells, and whereas the branches of the latter end in sensory nerve-endings in different parts of the body, the processes of the cells of type XI. end in nerve-endings in the ganglion or adjacent nerve trunk. The nerve-endings are modified Vater-Pacinian bodies, end-plates, and different forms of end-bulbs. The presence of multipolar cells of this variety in the normal ganglion is of importance in revealing possible sources of error in work like that of Nageotte, who described similar cells in transplanted ganglia which he believed to be an attempt on the part of the normal cells to regenerate.

A chapter is devoted to the description of other nerve fibres which enter the spinal ganglia and end in them. Sympathetic nerves end in the pericellular network of all types of ganglion cells. Other fibres, medullated and non-medullated, and probably of cerebro-spinal origin, enter the ganglia and end in relation to the ganglion cells or their processes.

The different types of cells are considered by Dogiel to

indicate differences in function, but what physiological rôle each type subserves is not determined. The work marks a large advance in our knowledge of the anatomy of the ganglion cells, and will stimulate fresh interest in the subject. A wide field for research is opened up, and new ground is afforded for the investigation of the different paths of afferent impulses to the central nervous system.

A special word of praise must be given to the plates illustrating the book; they are numerous, admirably executed, and are works of art in themselves.

PERCY T. HERRING.

RESEMBLANCES BETWEEN THE HUMAN SPINAL CORD AND (398) THAT OF ANIMALS. (*Zur Tierähnlichkeit im menschlichen Rückenmarke.*) PAUL BIACH, *Neurol. Centralbl.*, Juni 1, 1908, p. 507.

THE writer describes abnormal appearances which he has met with in human spinal cords which resemble the structure of the cord in animals. In some cords the substantia gelatinosa which normally encloses the posterior horn somewhat like a gothic arch shows two well-marked convolutions and merely lies on the top of the grey matter without surrounding it. Again, the grey matter is unusually rich in fibres; indeed, so numerous are these fibres, coming from the substantia reticularis or posterior columns, as in the horse and pig, and so far forward are they situated, that the posterior horn is sometimes completely cut off from the grey matter of the anterior horn. These changes are limited to the cervical region. An abnormality of the posterior roots almost always co-exists, the glia in the posterior horn extending out into the posterior root for some little distance. Bauer has shown that this is the normal condition in a large series of mammals, and he has also described it in cervical tabes in man. Levi, on the other hand, has shown that in the human subject the transition between glia and the connective tissue of the root takes place normally within the cord in the cervical region.

Among 27 pathological cords examined by the author, in which one or other of these abnormalities was present, in 16 both of the appearances above described were met with. It is argued that this coincidence must be more than mere chance. The author examined in all 50 pathological and 14 normal cords. In 27 of the pathological cords, one or both of the abnormalities were present, while in only 1 of the normal cords was this so. Among 17 cases of tabes, in only 4 were these abnormal features absent. The author concludes finally that a cord, which in its features resembles that of lower animals, is especially predisposed to tabes.

EDWIN BRAMWELL.

- ON THE RELATIONS BETWEEN THE ARCUATE NUCLEI AND**
 (399) **THE EXTERNAL ANTERIOR ARCIFORM FIBRES OF**
THE MEDULLA OBLONGATA. (Sui rapporti tra i nuclei
 arcuati e le fibre arciformi esterne anteriori della midolla
 allungata.) VOLPI-GHIRARDINI, *Riv. Ital. di Neuropatol., Psych.*
ed Elettroter., June 1908, p. 266.

THE author finds that the arciform nuclei are peculiar to man, although there are occasionally rudimentary traces of them to be found in the chimpanzee, and he associates this development of the nuclei with the function of the maintenance of equilibrium. He thinks that the external arcuate fibres enter into connection with the arciform nuclei—the occasional concomitant variation in the numbers of the fibres and of the nuclei substantiating this—and that the more ventral of the arcuate fibres are specially associated with the arciform nuclear cells. It is certain, however, that not all the arciform fibres are derived from the arcuate nuclei, as these fibres may be developed although such nuclei are absent.

ALEXANDER BRUCE.

- ON THE STRUCTURE OF NERVE CELLS OF THE ELECTRIC**
 (400) **LOBE AND OF THE NERVE ENDINGS IN THE ELECTRIC**
ORGAN OF TORPEDO OCELLATA. (Sur la structure des
 cellules nerveuses du lobe électrique, et des terminaisons
 nerveuses dans l'organe électrique du *Torpedo ocellata*.) G.
 PIGHINI, *Anat. Anz.*, xxxii. Band, No. 19-20, 1908.

PIGHINI has used Cajal's reduced silver method in his studies. The nerve cells show numerous dendrites, each of them composed of a small bundle of fibrils which, on arriving at the periphery of the cell, spread out fan-wise, the different fibrils passing in all directions. Two kinds of fibrils are seen—stout and fine. The stoutest fibrils stretch out quite to the periphery, where they interlace with other stout fibrils from neighbouring dendrites. From these are given off the finest fibrils, which pass obliquely towards the centre of the cell, interlacing with each other and forming small polygonal spaces, the whole of which has the appearance of a network. This network is distributed all over the cell body, and becomes denser in the interior. Arriving at the oval central space which contains the nucleus, the fibrillar meshwork becomes much thicker, and inserts itself, or is continued into a network of much stouter fibrils which envelop the nucleus. This perinuclear network is composed of fibrils similar to the peripheral fibrils; they assemble together and unite into a bundle directed towards the periphery at precisely the place where the axis-cylinder

arises, with which it is continuous. He only observed one axis-cylinder to each cell (Cantani states that there are several), which, at a short distance from its origin, narrows down abruptly and is directed towards that pole of the lobe where all the fibres reunite in the nerve which passes to the electrical muscles.

The dendrites, according to him, *only* contract relations of *continuity* between different cells; he has never seen free terminations of fibres or fibrils. He describes this method of junction as follows:—a dendrite from one cell arrives at the surface of a neighbouring cell, generally in a different plane, and divides into several fibrillar bands, which in turn break up into their component fibres and contribute to the formation of the peripheral reticular layer of stout fibrils previously mentioned. In some cases he observed two cells in the same plane and very near one another, exchanging between them many fine dendrites.

With regard to the manner of the termination of the nerves in the muscles of the electrical organ, he finds, as Retzius previously found, that they ramify dichotomously and terminate freely, and do not form a network.

JOHN TURNER.

PHYSIOLOGY.

THE FATIGUABILITY OF NERVES. (Über die Ermüdbarkeit des (401) Nerven.) A. BECK, *Pflüger's Arch.*, Bd. cxxii., H. 12, 1908, p. 585.

THIS investigator studied the effect of prolonged stimulation of the cervical sympathetic upon the iris of the eye of the cat. The experiments continued for several hours, seventeen in one case, with short intervals every hour or so of less than two minutes, the continuous stimulation by rapid induction shocks being of rather greater strength than was sufficient to give a maximal dilatation of the pupil. No effects were observed which might be attributed to fatigue of the nerve, and the author agrees with the older conclusions of Bernstein and others.

T. GRAHAM BROWN.

THE LOSS OF CONDUCTIVITY OF MOTOR AND SENSORY (402) NERVES IN THE FROG AT INCREASED TEMPERATURES. (Erlischt Leitungsvermögen motorischer und sensibler Froschnerven bei derselben Temperaturerhöhung?) MAX HAFEMANN, *Pflüger's Arch.*, Bd. cxxii., H. 10 and 11, 1908, p. 484.

THE author of this paper investigated the effect of heat on the conductivity of motor and sensory fibres in the sciatic nerve of

the frog. He severed one leg, in the decapitated frog, above the knee, leaving only the sciatic nerve in continuity with the body, and he applied heat to this part of the nerve, and stimulated it above and below the heated portion. The contraction of the muscles of the severed leg served to give an index for the conductivity of the motor fibres when the nerve was stimulated above the heated part, while the contraction of the muscles of the opposite leg served as an index of the conductivity of the sensory fibres when the nerve was stimulated below this point. It was found that, if the nerve was heated by means of hot Ringer's solution, to a temperature of 50° C., the conductivity of both motor and sensory fibres disappeared in a few seconds; on the other hand, the temperature of 42° C. did not disturb the conductivity within twenty minutes. Within these two limits of temperature the conductivity remained for a shorter or a longer time before it disappeared. The sensory fibres were always the first to be paralysed.

T. GRAHAM BROWN.

CONTRACTION OF FROG'S MUSCLE IN STRYCHNINE POISON-

(403) **ING.** (*Beiträge zur Kenntnis der willkürlichen Bewegung. I. Die Kontraktion des Froschmuskels bei Strychninvergiftung.*) ADOLF BASLER, *Pflüger's Arch.*, Bd. cxii., H. 8 and 9, 1908, p. 380.

THE forms of the contraction curves of the frog's gastrocnemius muscle were studied in animals treated with 0.1 to 0.5 mgrm. of strychnine, the contractions being sometimes obtained by stimulation of the sciatic nerve electrically, sometimes caused only by the spasm of the animal. The forms of these two kinds of curve were very similar, but in the latter case were rather higher and longer than in the former. This stretching of the curve is to be ascribed to a change in the condition of the muscle. In the later stages of the poisoning the muscles did not react to voluntary stimuli, while they would do so to direct stimulation, a result of the curari-like effect of strychnine. The electrical excitability of the nerve disappears much sooner than the excitability to voluntary impulses. The electrical current of action was also investigated, and it was found that in the strychnine single spasms the negative variation had a course slower by $\frac{1}{2}$ second than in the case of muscle contractions obtained artificially; it was also higher.

T. GRAHAM BROWN.

AN EXPERIMENTAL INQUIRY INTO THE ACTION OF
 (404) **ADRENALIN ON THE CENTRAL NERVOUS SYSTEM**
OF THE RABBIT. (*Experimentelle untersuchungen über*
die Wirkung des Adrenalin auf das Zentralnervensystem des
Kaninchens.) R. SHIMA, *Arb. a. d. Neur. Inst. an der Wien.*
Univ. Obersteiner, Bd. 14, 1908.

IN continuation of the work of W. Erb and others, the author has investigated the action of adrenalin on the central nervous system, especially with a view to determining what effects are produced by acute and chronic adrenalin poisoning, and whether these effects are indirect and due to lesion of the vessels, or directly due to toxic action. Rabbits of different ages were used, and adrenalin injections in varying doses were made into the marginal vein of the ear. The animals lived from 1 to 132 days.

The author finds that adrenalin produces effects on the central nervous system, which he groups as (1) *localised*, due to degeneration of the vessels, and consisting of infiltration and hyperæmia of the pia mater, and of small areas of softening and hæmorrhage in the brain; (2) *diffuse*, characterised among others by changes in the "ganglion" cells of the whole cortex, which show shrinking, vacuolation of the cell body, and corkscrew appearance of the dendrites, by increase in the glial tissue, and by proliferation of the ependyma cells.

The diffuse changes are seen especially in chronic adrenalin poisoning, and the author believes, in opposition to prevailing views, that adrenalin has a direct toxic action on the central nervous system, apart from its effects on the blood vessels.

He points out that in many respects these morbid appearances produced by adrenalin resemble those seen in progressive paralysis; and, further, that they may be of importance in regard to the observations of Schur and Wiesel, who have suggested that the existence of adrenalin in the blood is the cause of the rise of blood pressure in nephritis, for the symptoms of uræmia may be due to chronic adrenalin poisoning.

He concludes that, of poisons formed in the body itself, adrenalin plays a more important part in reference to the central nervous system than has hitherto been supposed.

J. A. GUNN.

PSYCHOLOGY.

COLOURED THINKING. DAVID FRASER HARRIS (St Andrews), (405) *Journ. Abnorm. Psychol.*, June-July 1908, p. 97.

COLOURED THINKING is to be distinguished psychologically from coloured sensation. Coloured sensations are coupled sensations (synæsthesiæ) as when heard sounds arouse colours, thus certain persons have coloured hearing (audition colorée). To some people a high note has, *e.g.*, a blue or green colour, a low note a deep red or violet.

Monsieur Peillaube (*Rev. Phil.*, Paris, 1904) has examined the coloured hearing of four persons with a view to discovering the bond between the sensation of one kind—the tone, and the sensation of another kind—the colour. Locke's blind man, to whom scarlet was "like the sound of a trumpet," is an early case of this sort of thing.

Chromatic conception is somewhat different. Here it is the thought, the idea, that is possessed of colour. There are certain persons who habitually think of "things," words, etc., as coloured; to them the hours of the day, the days of the week, the months of the year, etc., have each a distinct colour. The coloured concept may be called a psychochrome (Galton).

People differ considerably in their power of visualising (exteriorising); Mr Francis Galton has classified them (1883) into strong visuals and poor visuals; only the former are likely to be capable of coloured thinking. The characteristics of coloured thinking may be thus summarised:—

1. The associations between the concept and the colour have been formed at a very early age. In all Mr Galton's cases and in all I have examined this is so. Mr E. S. Holden (*Nature*, 1891, Vol. xlv.) reports on the psychochromes of his daughter at the age of seven. "Ever since childhood I have always seen," "as far back as I can remember," are the phrases used.

2. In the next place we may note the total lack of agreement between the colours attached to the same concepts in the minds of seers.¹ To take a definite case, the thought of the vowel "u" is to one seer grey, to another yellow, to four others black, brown, blue, and green respectively.

3. The third characteristic of psychochromes is their extreme definiteness in the minds of seers. Contrary to what might be expected, the precise colours attached to concepts are by no means vague or beyond accurate description. Indeed, a seer is most fastidious in his choice of terms to give adequate expression to his chromatic imagery. One is not content with speaking of Sep-

¹ Mr Galton's term for coloured thinkers.

tember as grey, he calls it "steel-grey"; another speaks of a dull white, a silvery white, etc. The degree of chromatic precision which can be given by seers to the description of their visualisings is as surprising as anything else in this curious subject.

4. Though so distinct, psychochromes never attain to hallucinations; they have all the definiteness of a thought without the verisimilitude of a "subjective" sensation. Psychologically this is intelligible, since the psychochrome is not a sensation but a concept.

5. The next distinguishing feature is that this mental faculty is hereditary, "very hereditary" Mr Galton puts it. The tendency to coloured thinking is congenital, not environmentally produced; it is due to "Nature not nurture" (Galton). The same is true of coloured hearing. In one case, "Un cas héréditaire d'audition colorée" (Lauret et Duchassoy, Abst. in *Centralbl. f. Physiol.*, Leipzig and Wien, 1888), a father and son each associated both sounds and thoughts with colours. With vowels (sounds or thoughts) they associated "gay" colours, with the consonants shades of grey.

These cases were typical in that the father and the son did not associate the same colour with a particular vowel heard or thought of.

6. Perhaps the most marked characteristic of coloured concepts is their unchangeableness throughout life. Middle-aged persons will tell you there has been no modification of them since early childhood. Mr Galton remarked of them, "they are very little altered by the accidents of education." Just as their origination is not due to the influence of the environment, so the environment exercises no modifying influence on them as life proceeds.

The precise colours associated are those in the spectrum as well as white, black, grey, and a very large number of tints and shades.

The colours differ greatly in the relative frequency of their occurrence: of 100 psychochromes, white constituted 24, brown 24, black 17, yellow 11, green 7, blue 5, red 4, pink 3, cream 3, orange 1, purple 1 per cent.

The kind of people who are coloured thinkers are, according to Mr Galton, above rather than below the intellectual average. Coloured thinking occurs both in men and in women; as an example of the latter we may take Ellen Thornycroft Fowler, some of whose psychochromes are described in her novel, "In Subjection" (Hutchison & Co., 1906).

Can we arrive at any explanation of synæsthesia and of coloured thinking? As to coupled sensations, Monsieur Peillaube has attempted an analysis of its origin. He examined the case of

a Monsieur Ch——, to whom low notes (heard) called up violet, thus—

To this person low notes were “douce et profondes,”
the colour violet was “douce et profonde,”
therefore low notes were violet.

The link “douce et profonde” was arrived at only after a great deal of introspection. The sequence was $x-l-y$, where l was the emotional link which had long ago dropped out of consciousness, so that ever after x and y seemed instantaneously and indissolubly bound together.

It is quite possible that the concept and the colour in a psychochrome might in some cases have a similar origin. To take one example, where the concept “February” is always white, it is arrived at thus—

Snow is white,
the earliest February remembered was snowy,
therefore February is white.

While some psychochromes may be so explained, it seems hopeless on similar lines to explain such curious and arbitrary associations as October with black, Monday with yellow, Thursday with white, 9 P.M. with yellow, 11 P.M. with green, and finally the vowel “u” with white, yellow, black, brown, blue, green in the minds of six different persons respectively. My experience is that of Mr Galton: those questioned say, “I cannot account for their origin in any way.”

There seems no light to be thrown on coloured thinking by considering it in connection with any of the theories of colour vision. We have seen that psychochromes are remarkably stable throughout life. Those who are coloured thinkers are somewhat above the general intellectual average. Coloured thinking may thus be regarded as in a category allied to genius; genius is notoriously not conferred by training or education; if not inherited it cannot be acquired. These things show us that it is not in the ordinary type of mind, but in the recesses of the slightly aberrant, that the more recondite problems of mental physiology present themselves for solution.

AUTHOR'S ABSTRACT.

AN EXPERIMENTAL STUDY OF SLEEP. (Part II.) BORIS SIDIS,
(406) *Journ. of Abnorm. Psychol.*, June-July 1908, p. 63.

IN continuation of the account of his study of sleep, Dr Sidis here describes his experiments on cats, dogs, and children.

The kittens and puppies which were used in preference to older animals were usually wrapped in a cloth in such a way as to prevent movement, and conduce to the monotony required. Their eyes were then closed, and sleep quickly set in even in the case of

puppies which at first violently resisted the endeavour to restrict their activities. Slight catalepsy was frequently observed in the paws at the beginning and towards the end of the sleep-state, this being one of the signs of the presence of the hypnoidal or sub-waking state. This condition was more noticeable in the puppies than in the kittens, and in older than in younger animals. An interesting feature of the experiments was the habituation of the animals to the oncoming of the sleep state. With some of them it became sufficient to lay them on their sides and merely touch the eyelids.

In the case of the children the subjects ranged from infants a few days old to children twelve and thirteen years of age. In young babies, as Dr Sidis points out, consciousness is concerned not with external but with internal sensations. Hence if a baby is hungry, the monotony necessary for the induction of the sleep state cannot be produced merely by shutting the eyes and restraining the activity. Babies a few weeks old are easily put to sleep by enforcing monotony of sensation. Cataleptic conditions are frequently observed. In older children the hypnoidal states become more marked, and hypnosis, and even somnambulistic states sometimes appear. Thus "in infants and children, as in the lower animals, sleep, hypnosis, and hypnoidal states are intimately related, sleep presenting complex manifestations of sub-conscious states which become fully developed in the adult."

MARGARET DRUMMOND.

**EXPERIMENTS IN PSYCHO-GALVANIC REACTIONS FROM CO-
(407) CONSCIOUS (SUB-CONSCIOUS) IDEAS IN A CASE OF
MULTIPLE PERSONALITY. MORTON PRINCE and FREDERICK
PETERSON, *Journ. of Abnorm. Psychol.*, June-July 1908, p. 11.**

THIS paper describes experiments devised to obtain further evidence regarding the existence under certain conditions of conscious processes of the presence of which the subject is unaware. It has been shown by Tarchanoff, Veraguth, Jung, and others, that when a weak electric current is passed through the body, the current is increased whenever emotion is aroused. This fact was made use of in testing for the co-conscious processes. Dr Prince had a very complex case of multiple personality, in which one of the personalities claimed to be co-conscious all the time that another was in the ascendancy. Eight sets of experiments arranged with a view to testing this claim are described. The guiding principle of these may be thus indicated. A series of words was selected in which some occurred which roused vivid emotions in the subordinate personality, but which had no special

association for the personality dominant at the time. If the subordinate personality were really present and active, then it was thought that the galvanometer should indicate the rise of emotion when the test words were pronounced. For example the word "Smith" recalled an episode in the life of personality B of which personality A had no knowledge; of this episode B was now ashamed. The word "Smith" was introduced into a series of indifferent words and the current passed through A. A marked rise took place at the test word. This rise Dr Prince attributes to the co-conscious influence of B. Tracings are given representing various tests of like nature, in all of which the emotions of the co-conscious subordinate personality seem to be able to produce an effect on the galvanometer.

MARGARET DRUMMOND.

PATHOLOGY.

SOME LESIONS OF THE SPINAL CORD PRODUCED BY EXPERIMENTAL CAISSON DISEASE. A. E. BOYCOTT and G. C. C. DAMANT, *Journ Path. and Bact.*, Vol. xii., 1908, p. 507.

As the result of the examination of a series of goats dying of caisson disease, or killed after exposure to compressed air, the authors show that the distribution of the lesions found in the spinal cord follows theoretical considerations. (1) Bubbles are not uncommon outside the vessels in the substance of the cord, their frequency here being doubtless due to the fatty nature of the myelin. They are, however, about five times more abundant in the white than in the grey matter; the fatty content of the two parts being about the same, this distribution must be due to the difference in blood supply, the dissolved gas being carried away by the blood much more easily from the grey than from the white matter. In the same way, the bubbles are more frequent in those parts of the cord which have an abundant (lumbar and cervical enlargements) than in those which have an indifferent (lower dorsal) blood supply. (2) The paralysis which often follows exposure to compressed air is nearly always a paraplegia due to softening of the white matter in the dorsal cord. In the authors' experience the lesion, due to air embolism, is strictly confined to the white matter, and is either restricted to, or much more marked, in the dorsal cord than elsewhere. This distribution is explicable on the basis of the varying activity of the circulation in different parts of the cord. In the central nervous system above the cord, neither extra-vascular bubbles nor softening was found in any case.

AUTHOR'S ABSTRACT.

ON THE POWER OF CHOLESTERIN AND NERVOUS SUB-
(409) STANCE TO NEUTRALISE THE HÆMOLYTIC ACTION
OF LECITHIN AND SPECIFIC SERUMS, G. PIGHINI,
Riv. Sper. di Fren., Vol. xxxiv., fasc. I.-II., 1908.

PIGHINI by a series of experiments shows that cholesterin and nervous tissue of brain or cord (ox, dog) exert a neutralising effect on the alleged hæmolytic power of lecithin and specific serums. This action seems to be in proportion to the quantity of the substance used in the reaction, since one can see that increased quantities of lecithin or serum require increased doses respectively of cholesterin or nervous tissue emulsion to prevent hæmolysis.

He believes that the neutralising substance in both cases is cholesterin, which occurs free in the central nervous system. In discussing its mode of action he refers to the views of Neuberg, Reicher and Rosenberg, according to which many hæmolytic processes are included among the lipolytic, and which attribute to the fatty material of the red corpuscles the essential part in the production of specific hæmolysins. If these views are confirmed he believes that it will be possible to trace a connection between the lipolytic action and the antagonistic action of cholesterin.

He refers to the experiments of Wassermann and Bruck on the prevention of the hæmolysis ordinarily produced by an incubated hæmolytic serum and its complement, when there is added an extract of a syphilitic organ (liver), and a certain quantity of cerebro-spinal fluid containing the anti-bodies sought for, and which they have interpreted as a "deviation of the complement," *i.e.* the complement, instead of effecting hæmolysis, helps the reaction between the specific toxin and the supposititious anti-toxin.

Wassermann, in consequence of the repeated positive results he and others have obtained, regards the syphilitic nature of tabes and general paralysis as proved. But the experiments of Levaditi and Jamanouchi throw grave doubts on the accuracy of Wassermann's conclusion. They showed that the active principle of extract of syphilitic liver is contained in the alcoholic extract, and is composed of a mixture of fats and bile acids, and also that the active principle of the serum of syphilitics and the cerebro-spinal fluid of general paralytics is equally extracted by alcohol and consists of fats and salts; and this alcoholic extract hinders hæmolysis in the presence of a watery or alcoholic extract of liver, and even in the absence of liver extract, provided only that the dose is stronger. They find that if one compares the anti-complementary strength of extracts of serums or pathological fluids with extracts from normal persons, there is only a small quantitative difference between the two. According to them it is not correct to speak of

deviations of complements brought about by true antigens and antibodies. It is a question rather of substances soluble in alcohol and ether, which are found in larger quantities in pathological tissues and fluids.

In view of Pighini's results it will be necessary to ascertain if the alcoholic extracts obtained by Levaditi and Jamanouchi contain cholesterin, which occurs free in bile and nervous tissues. An alcoholic extract of liver may contain cholesterin, and if this body is not usually a component of cerebro-spinal fluid, it may escape into it from the surrounding nerve substance in pathological conditions.

JOHN TURNER.

CLINICAL NEUROLOGY.

AMYOTONIA CONGENITA. JAMES COLLIER and S. A. K. WILSON, (410) *Brain*, 1908, p. 1.

IN this paper the authors have collected all the hitherto recorded cases of this condition, amounting to twenty-one. In addition, they report four new cases, making a total of twenty-five cases described and commented on. The material is sufficient to make a clinical description reliable, although pathological information is at present limited to the somewhat contradictory results obtained in two cases.

Amyotonia congenita (which is preferable to myatonia congenita) may briefly be defined as a condition of extreme flaccidity of the muscles associated with an entire loss of the deep reflexes, most marked at the time of birth, and always showing a tendency to slow and progressive amelioration. There is great weakness, but no absolute paralysis of any muscle. The limbs are most affected, the face is almost always exempt. The muscles are small and soft, but there is no local muscular wasting. Contractures are prone to occur in the course of time. The faradic excitability in the muscles is lowered, and strong faradic stimuli are borne without complaint. No other symptoms indicative of lesions of the nervous system occur.

In nearly all cases the paralysis has been obvious at the time of birth, or it has been noticed so few hours after birth as to make it certain that the condition has been pre-natal. In four out of the twenty-one, however, it seems clear that the weakness either supervened or became much worse some months after birth.

The affection is always strictly symmetrical upon the two sides; it may be universal in distribution, but the muscles of mastication and deglutition seem always to have escaped. When the affection is general the several parts of the body are never equally affected; the lower extremities are most often and most deeply involved,

next the upper extremities, then the trunk, and lastly the face. The distribution of the affection is not in terms of the long axis of the body.

In the limbs the amyotonia may be distributed equally upon both proximal and distal parts, but this is unusual. It has been stated by most of the previous writers upon this subject that the proximal muscles are always more severely affected, but it seems to us certain that this conclusion has been arrived at from the obvious disadvantage at which the proximal muscles act upon the limbs against gravity as long levers. There is no local muscular atrophy comparable to the local atrophy which is characteristic of all cases belonging to the group of the myopathies. Contractures have only been met with in the lower extremities, and they do not correspond either in position or in degree with the severity of the amyotonia.

This tonelessness of the affected muscles is the most striking feature in the condition. However complete the apparent paralysis may be, yet every muscle when put into a favourable position as regards work involved, contracts voluntarily. The relaxation of muscles and ligaments allows of the most remarkable over-extension of the joints, and leads to a highly characteristic flail-like condition of the joints when shaken.

Amyotonia congenita must be distinguished from acute infantile spinal muscular atrophy of familial type, a condition of complete atrophic palsy of the muscles with complete loss of faradic excitability, and of sensibility to all forms of stimulation in the region affected.

Whether amyotonia is a variety of myopathy, or whether it is a clinical and pathological entity entirely separate from the myopathies, is a matter of importance and difficulty. So far as the pathological evidence derived from the two cases which have been examined goes, it is quite compatible with a condition of myopathy, though the results in the two cases are widely at variance one with another. The following points are those on which the opinion is based, that amyotonia is, clinically at least, a distinct condition:—

1. The myopathies are conspicuously familial diseases, whereas no familial tendency has been recorded in amyotonia.
2. The several types of myopathy often show familial relationship one with another, whereas no case of amyotonia has been reported in a myopathic family.
3. A large majority of the cases of amyotonia are congenital, the condition being obvious at birth. In none of its several types is myopathy apparent at birth.
4. The characteristic muscular flaccidity of amyotonia is not present in myopathy.

5. The local muscular wasting that is a marked feature of myopathy is not present in amyotonia.
6. The course of myopathy is one of progressive increase of the muscular weakness, that of amyotonia is one of progressive amelioration of the symptoms.
7. Return of the deep reflexes after their persistent absence for months or years has been recorded several times in amyotonia, and has occurred in two of our cases under our observation. Such a return of an absent deep reflex has never been recorded in myopathy.

AUTHORS' ABSTRACT.

ARE THERE "FORMES FRUSTES" OR RUDIMENTARY FORMS (411) OF MUSCULAR DYSTROPHY (ERB), AND ARE THEY CURABLE? (Gibt es Formes frustes oder rudimentäre Formen des muskulären Dystrophie (Erb), und ist deren Heilung möglich? MARINA, *Deutsche med. Wochenschr.*, 1908, p. 1087.

IN this lecture, delivered by Marina at the Italian Congress of Pediatrics at Padua in October 1907, the question of the existence of rudimentary forms of muscular dystrophy is discussed.

Under the general heading of muscular dystrophy Erb included an infantile or pseudo-hypertrophic type, with or without implication of the face (Landouzy and Dejerine), a juvenile scapulo-humeral type, and a hereditary type. Such a disease, affecting the muscles in different degrees, and advancing often with great slowness, readily lends itself to classification into sub-varieties. It is well known that the pseudo-hypertrophy and the atrophy are often irregular and atypical, and that fibrillary tremors may even be observed in some cases, so as to give rise to uncertainty in the diagnosis; moreover, the course of the malady may be so slow as to cause little or no interference with muscular activity.

Marina gives an account of a mild case of juvenile muscular dystrophy in a young man aged 31, of feeble character and physique, who had a degree of atrophy (not enlargement) of the *supra-* and *infra-spinati* and of the *serrati*, more marked in the left side. The calf muscles were somewhat enlarged, and the patient climbed up his legs from the recumbent posture in a typical manner. Marina also recalls the occurrence of so-called "abortive" forms of muscular dystrophy (Oppenheim), in which the disease remains confined to the shoulder-girdle and does not tend to spread.

An account is given of another case, which he regards as a rudimentary type or "*forme fruste*," differing in certain particulars from the foregoing "abortive" type. The patient was a

girl, who originally came under observation at the age of 8½ for commencing scoliosis. The right shoulder was higher than the left, the scapulæ were winged at their lower angles. The *supra-spinati* were slightly atrophied, the *infra-spinati* slightly enlarged. The pectoral and deltoids and certain other scapular muscles were moderately wasted, whilst the right facial muscles were distinctly enlarged. The lower limb appeared normal, but the faradic excitability of the muscles was diminished. All the deep reflexes of the upper limbs were absent; those of the lower limbs were normal. The patient was muscularly feeble and easily tired. The patient was re-examined at the age of 14. By this time she had improved considerably in general muscular strength. A mild scoliosis was still present, but the general musculature appeared normal save for slight hypertrophy of the lower halves of the *infra-spinati* and diminished development of the muscles of the right upper arm. The right half of the face was still hypertrophied. Electrical excitability of the muscles was still below normal, and in the right hand-muscles no motion was obtained. In this case, therefore, the malady had apparently come to a standstill, and had even improved. Marina admits, however, that this apparent "cure" may prove to be but a period of remission or standstill in a disease which may later reassert its progressive nature.

PURVES STEWART.

THE DIFFERENCE BETWEEN CENTRAL AND PERIPHERAL
 (412) **FACIAL PARALYSIS.** (Die Unterschiede centraler und peripherer Facialis-lähmungen und die anatomische Grundlage derselben.) C. HUDOVERNIG, *Neurolog. Centralbl.*, 1908, p. 577.

THE essential clinical differences between facial paralysis of central and of peripheral type are commonly stated to be three in number. Firstly, in central facial paralysis the upper facial muscles are completely, or almost completely, spared, whereas in peripheral cases all the muscles, upper and lower, are affected. Secondly, electrical reactions of degeneration are absent in central and present in all moderately severe peripheral cases. Thirdly, it is stated that certain reflexes which are lost in peripheral cases are preserved in central paralysis.

With regard to the escape of the upper facial muscles in most of the central cases, the classic explanation of Charcot was that there exists in each hemisphere a common centre for the bilateral innervation of the upper facial muscles, so that when one centre is paralysed the other maintains its function. Hudovernig, however, holds that the true explanation is to be found in the fact that the upper facial muscles have a separate cortical centre from

the lower, and that the central path of the upper facial muscles is separate from the lower facial path in the internal capsule, hence it escapes in the usual central lesion. He also maintains that in the bulbar facial nucleus itself there is a dorsal portion corresponding to the upper facial muscles and a ventral corresponding to the lower muscles. Once the infra-nuclear course of the facial path is interrupted, these two divisions are implicated simultaneously, and no difference is possible between the degree of paralysis of upper and of lower muscles.

The remarks on electrical reactions contain nothing new. With regard to the third point—the alleged loss of the “supra-orbital reflex” in peripheral lesions, as compared with its preservation in central lesions—Hudovernig maintains that the supra-orbital phenomenon is not a true reflex, but simply a diffusion of mechanical excitability by direct stimulation of muscles and fasciæ. Moreover, the supra-orbital phenomenon can still be elicited after division of the trigeminal nerve or extirpation of the Gasserian ganglion, *i.e.* when the sensory limb of the supposed reflex arc is absent. It is, therefore, of no value in distinguishing central from peripheral lesions.

PURVES STEWART.

OPTIC AND OCULOMOTOR NEURITIS FOLLOWING GONOR-
(413) **RHCEA.** (Nevrite ottica e nevrite dell' oculomotore comune da intossicazione gonococcica.) F. BARILE, *Giornale di med. militare*, 1908, p. 275.

A SOLDIER contracted gonorrhœa in December 1905. In January 1906 bilateral gonorrhœal conjunctivitis developed, with predominance of the lesions on the conjunctival surface of the left upper lid. The conjunctivitis cleared up under treatment, but in the course of the year frequent relapses occurred. Ptosis, convergent strabismus and amblyopia developed in the left eye, and the reaction to light and accommodation in the left pupil was lost. The patient finally recovered a certain degree of vision in the left eye, but when last seen he had still some ptosis, strabismus and mydriasis.

J. D. ROLLESTON.

A CASE OF POLIOMYELITIS POSTERIOR OF THE GENICU-
(414) **LATE GANGLION.** (Ein Fall von Poliomyelitis posterior des Ganglion geniculi; anschliessend Betrachtungen über den dabei festgestellten Symptomenkomplex.) J. RAMSAY HUNT, *Neurol. Centralbl.*, Juni 1, 1908, p. 514.

THE symptoms of an herpetic inflammation of the geniculate ganglion are pain in the ear, an herpetic eruption on the concha

the external auditory meatus, and the tympanic membrane. Facial paralysis, deafness, and the Menière symptom-complex occur when there is an extension to the neighbouring nerve elements. When the inflammatory process is limited to the geniculate, the herpetic vesicles may be distributed on the drum, the external auditory meatus, the concha, the tragus, the anti-tragus, and its immediate neighbourhood. The author describes four types.

1. Herpes oticus characterised by pain in the ear, with an herpetic eruption in the distribution above mentioned.

2. Herpes oticus, herpes facialis, or herpes occipito-cervicalis, together with facial paralysis. In the latter case the herpes is distributed in the areas supplied by the 1st, 2nd, and 3rd cervical roots.

3. Herpes zoster involving one or more zones of the head, facial paralysis and diminution of hearing.

4. Herpes zoster of the head with facial paralysis and the Menière symptom-complex.

The Gasserian ganglion, the geniculate ganglion, and the upper cervical ganglia are liable to be affected together, as is the case with the spinal ganglia. Dr Hunt has collected 61 cases of herpes of the head with facial paralysis. In 20 there were distinct signs of involvement of hearing, six showing a mere diminution of hearing, while 11 exhibited in addition the Menière symptom-complex.

EDWIN BRAMWELL.

CHRONIC NEURITIS OF THE ULNAR NERVE DUE TO (415) DEFORMITY IN THE REGION OF THE ELBOW JOINT.

SHERREN, *Edin. Med. Journ.*, June 1908, p. 500.

ATTENTION is drawn in this paper to a condition which has often led to errors in diagnosis. The twenty-one previously recorded cases are tabulated and reviewed, and two upon which the writer operated fully recorded.

In most of the cases the deformity was a cubitus valgus, due usually to injury in early life, a fracture or epiphyseal separation at the lower end of the humerus. The interval between the injury and the onset of symptoms was found to have varied from six to thirty-six years. In all in whom a note on the condition of the nerve was made, it was found enlarged in a spindle-shaped manner.

The writer concluded that the neuritis was due to pressure upon or irritation of the nerve in movements of the elbow owing to its altered bony relationships.

The symptoms and treatment are shortly discussed.

AUTHOR'S ABSTRACT.

ON A CASE OF AMYOTROPHIC POLYNEURITIS. (A propos (416) d'un cas de polynévrite amyotrophique.) G. CATOLA, *Nouv. Icon. de la Salpêtrière*, 1908, p. 129.

THE patient whose case is here recorded was a young girl of 18 years, suffering from dementia præcox. During the course of a pulmonary and intestinal tuberculosis the patient developed a progressive amyotrophic paralysis, beginning in the upper limbs. Both paralysis and muscular atrophy were localised at the start to the muscles supplied by the musculo-spiral nerves, and in the lower limbs to the tendons of the external popliteal nerves; later the affection spread to the other muscles of the limbs and to the thoracic muscles. The muscular wasting was accompanied by loss of reflexes and R.D. No notable changes were detected in sensation. The cranial nerves and sphincters were intact.

The chief diagnostic difficulty in this case was its diagnosis from acute anterior poliomyelitis: the initial asymmetry of the disease and the practical absence of sensory phenomena would have been compatible with a poliomyelitis, but the strictly peripheral distribution of the muscular wasting and paralysis were in favour of a polyneuritis, so also were the picking-out of certain nerve-territories, and the tenderness on pressure of the nerve-trunks. The slow development of the disease, taking several weeks to attain its maximum, was also against a diagnosis of poliomyelitis.

Histological examination showed that the anterior and posterior roots of the spinal nerves were normal. The spinal cord, however, showed, by the Marchi method, well-marked diffuse discoloration of all the white matter — not the black globules characteristic of degeneration: evidently the myelin had undergone some chemical change. The peripheral nerves, on the other hand, showed degeneration of the axis cylinders and medullary sheaths, closely resembling the changes of Wallerian degeneration. This fact would tend to indicate that there is no fundamental difference between so-called "degenerative" and "secondary" neuritis. The integrity of the spinal nerve-roots is an interesting point to which special attention is called. The peripheral nerve fibres, therefore, were specially picked out by the tuberculous toxin, which spared the central nervous system. PURVES STEWART.

THE VALUE OF THE UNILATERAL LOSS OF THE KNEE JERK
(417) **IN THE DIAGNOSIS OF TABES.** (La valeur de la perte unilatérale du réflexe rotulien dans le diagnostic du tabes.)
R. BURNAND, *Rev. méd. de la Suisse Romande*, April 20, 1908, p. 282.

A SHOEMAKER, aged 57 years, was admitted to hospital on August 13, 1907, for attacks of vomiting and abdominal pain, which on

examination proved to be of the nature of gastric crises. The right knee jerk was completely absent, while the left knee jerk and both the tendo Achillis jerks were normal. The pupils were equal, and reacted to light. There was no Romberg's sign, ataxia, nor any anomaly of gait. Precipitate micturition and occasional delay in starting the act were also present. Syphilis was at first denied, but later the patient admitted that he had had a chancre on the penis at the age of 22 years. Lumbar puncture showed an abundant lymphocytosis, and was followed by headache and inequality of the pupils, the pupillary reflexes remaining normal. No further gastric crises occurred. Shortly before leaving hospital he suffered from lightning pains.

After a review of the literature the author concludes that unilateral abolition of the reflexes is not very exceptional in tabes, and attributes it to asymmetry of the lesions. Absence of the knee jerks coinciding with persistence of the Achillis jerks is more rare. Cases of the kind have been reported by Babinski, Camus and Sézary, Kollarits and Berger.

The unilateral loss of the knee jerk in this case is attributed to local traumatism. For the last twenty years the patient had been in the habit of mending boots on his right knee. That trauma of this kind in the absence of tabes does not affect the knee jerk was proved by the fact that subsequent examination of about twenty shoemakers who had been using the same knee for the last fifty years showed a perfect equality of the knee jerks on both sides.

J. D. ROLLESTON.

ON A CASE OF SYPHILITIC SPINAL PARALYSIS. (Über (418) einen Fall von syphilitisches Spinalparalyse.) RENNER, *Deutsch. Zeitsch. f. Nervenheilk.*, 1908, p. 451.

THE case here recorded was that of a man aged 36, the date of whose syphilitic infection was uncertain. For nine years before his terminal illness he complained of pains in the legs and cramps in the calves; four months before his death definite spastic phenomena developed, together with unsteadiness of gait. A course of mercurial inunction was without effect, the gait became much more unsteady, and seven weeks before death bladder trouble developed. On admission to hospital he had definite evidences of cystitis. There was loss of pupillary light reflex, optic atrophy, ataxy of the upper limbs, a zone of slight diminution of tactile sensibility around the upper part of the abdomen—phenomena suggestive of tabes; but, on the other hand, the spastic paresis of the lower limbs, the brisk deep reflexes and the presence of extensor plantar reflexes showed that the pyra-

midal tracts were also implicated. Towards the end psychical symptoms appeared, apparently the result of toxic absorption from the bladder infection.

Autopsy confirmed the diagnosis of syphilitic combined system degeneration. The changes in the cervical cord corresponded with those in an incipient tabes superior. They implicated mainly the postero-external columns, and were most marked in the upper thoracic and cervical regions, increasing in intensity as they were traced upward. In addition, there was well-marked pyramidal degeneration, increasing in intensity on tracing downward. There was no thickening of the pia, nor any local meningitis or compression. There is a good plate showing the degeneration in the cord.

PURVES STEWART.

OBSERVATIONS ON CEREBRO-SPINAL MENINGITIS. STUART
(419) M'DONALD, *Journ. Pathol. and Bacteriol.*, Vol. xii., No. 4, April 1908, p. 442.

THIS paper deals with (1) observations on the staining reactions of the meningococcus, (2) the presence of leptothrix forms in the cerebro-spinal fluid in cases of acute meningitis, (3) some experimental results in meningococcal infection.

I. *Staining Reactions of the Meningococcus.*—The author is of opinion that at certain times certain stains of undoubted meningococci show a variable reaction towards Gram's stains. In over fifty cases of epidemic meningitis personally studied, meningococci were demonstrated forty-three times in the cerebro-spinal fluid during life or in the meninges at the post-mortem examination.

In thirty-five of these cases a culture of the meningococcus was obtained, but only in three of the strains was the Gram variable reaction observed. In none of the cases was a Gram positive reaction on the part of the organism demonstrated in the cerebro-spinal fluid or in the tissues, but on several occasions meningococci were seen which, after staining with Gram's gentian violet solution, and the application of the iodine solution, resisted decolorisation with absolute alcohol for one minute. The three strains referred to were cultivated from the cerebro-spinal fluid during life in typical acute cases. In all of the three strains Gram positive forms appeared on cultures or sub-cultures; in no case did the organism grow on gelatine at room temperature; in two of the strains at least the author considers that the possibility of two organisms being present was absolutely excluded. In the case of the third strain, from a very acute fatal case, the organism was typically Gram negative in the cerebro-spinal fluid; but in the first

sub-culture some Gram positive cocci appeared. In the case of some of the diplococci one element was Gram positive, the other Gram negative. An intraperitoneal injection in a mouse of five minims of an emulsion of a twenty-four hours' culture of the organism on serum agar proved fatal in less than twenty hours. The organisms found in the peritoneal exudate were morphologically identical with meningococci. The organism was recovered in fine culture from the peritoneum, and films from this culture showed the organism to be still Gram negative.

II. *The Presence of Leptothrix Forms in Cerebro-spinal Fluid in Acute Cases of Meningitis.*—In five cases of acute meningitis Gram negative bacilli have been observed in the cerebro-spinal fluid during life, in two cases no other organisms could be demonstrated, in other two cases Gram positive diplococci were present together with the bacilli; in the fifth case an organism resembling a true meningococcus was present. The bacilli varied much in length, being sometimes seen in a short diplo-bacillary form; at other times long thread-like forms were present, staining faintly and irregularly. The author believes the organism to have been the same in all the cases. In one case a pure culture of the bacillus was obtained and proved to be a leptothrix form, long sinuous filaments appearing on blood agar. No proof of pathogenic property on the part of the organism was obtained. The significance of this leptothrix invasion is not quite clear; it may be a secondary infection, but on the other hand, in the two cases in which a post-mortem examination was obtained, the organism appeared to have at least modified the infection, as the distribution of the exudate in the meninges was vertical rather than basal, and the exudate itself was more fibrinous than is usually seen in meningococcal meningitis.

III. *Some Experimental Results in Meningococcal Infection.*—The animals employed were mice, guinea-pigs, monkeys, and a goat. Of the smaller animals mice were found most suitable for experimental work. Intraperitoneal inoculation of cultures of the meningococcus or of cerebro-spinal fluid containing the organism gave fairly constant results, the animals usually dying in from twelve to thirty hours. Guinea-pigs, even when young, did not prove suitable subjects, showing apparently a greater resistance to the meningococcus. A goat, given an intra-dural (spinal) injection of thirty minims of cerebro-spinal fluid from an acute case, containing numerous meningococci, suffered no inconvenience whatever. The author confirms Flexner's experimental observations on monkeys, and by using cerebro-spinal fluid, containing meningococci, rather than cultures, has obtained positive results with much smaller doses of meningococci than Flexner employed. Some experiments have been performed with the object of throw-

ing light on the natural paths of infection by the meningococcus. One monkey was inoculated intravenously with fifteen minims of cerebro-spinal fluid from an acute epidemic case. The animal died on the fourth day with symptoms of acute toxæmia; there were no meningitic symptoms. Post-mortem, no meningitis was found, but there were evidences of an acute general toxæmia. In another experiment fifteen minims of an emulsion of a twenty-four hours' culture of a meningococcus were injected into the sheath of the sciatic nerve, the object of the experiment being to determine the possibility of infection of the cord by way of the perineural lymphatics. The animal showed no symptoms for between three and four weeks, but died rather unexpectedly just over four weeks from the time of injection. Post-mortem, no apparent cause for death was found. There was no meningitis. No meningococci could be demonstrated in the tissues, or recovered by culture. Locally at the point of inoculation there was chronic inflammatory change in the sciatic itself and in its sheath. In order to determine the possibility of a primary intestinal route of infection, cerebro-spinal fluid from an acute meningococcal case was injected into the jejunum of a monkey after abdominal section. The animal almost at once showed symptoms of general toxæmia; there was slight diarrhoea, but no indications of meningitis; death occurred on the fourth day. Post-mortem, there was found no peritonitis and no meningitis, but there was evidence of a general acute toxæmia. The mesenteric glands near the jejunum were enlarged and acutely inflamed, and Gram negative diplococci morphologically identical with meningococci were demonstrated in them; the organisms, however, could not be recovered in cultures.

The author believes that further experiments on similar lines may be expected to yield definite information as to the natural routes of infection, which cannot be regarded as satisfactorily established so far.

AUTHOR'S ABSTRACT.

TYPHOID BACILLI IN THE CEREBRO-SPINAL FLUID IN
(420) TYPHOID FEVER. (Ueber den Nachweis von Typhus-
 bazillen in der Cerebrospinalflüssigkeit bei Typhus abdomin-
 alis.) NIETER, *Münch. Med. Woch.*, 1908, No. 19, p. 1009.

THE diagnostic value of lumbar puncture in typhoid fever is well illustrated by the following case. A man, aged 20 years, died after a week's illness with symptoms of meningitis. The diagnosis lay between typhoid fever and epidemic cerebro-spinal meningitis. A complete autopsy was not performed, but cultures were taken from the nasal mucus, cerebro-spinal fluid, fæces and spleen. No meningococci were found in the nasal mucus, but typhoid bacilli

were present not only in cultures from the spleen and faeces, but also in that from the cerebro-spinal fluid, in removal of which special care had been taken to prevent contamination. Nieter alludes to other cases in which the diagnosis of typhoid fever was made during life by examination of the cerebro-spinal fluid.

J. D. ROLLESTON.

A CASE OF ACUTE POLIO-ENCEPHALOMYELITIS IN A BOY (421) OF THREE. (*Cas de Polio-encéphalomyélite aiguë chez un Garçon de trois ans.*) M. ACUÑA, *Arch. de méd. des Enf.*, Juin 1908, p. 405.

THIS boy had some slight fever, diarrhoea, and an impetiginous eruption of the skin for twelve days, then his legs became paralysed; the paralysis rapidly ascended till in a few days there was complete paralysis of the limbs, trunk, neck, and palate. Death with bulbar symptoms twenty days from the onset. The pathological changes in the central nervous system were mainly seen in the blood vessels. These were much distended, some thrombosed, but most in evidence was the peri-vascular infiltration of small round cells. This reaction was not confined to the anterior cornua, the posterior cornual vessels being also considerably affected, and also those of the nerve roots and pia-arachnoid. Similar more scattered areas were also found in the medulla, pons and basal ganglia. The dorsal cord was most affected. The nerve cells showed only slight chromatolysis. The author attributes the disease to some virus carried by the blood-stream. No organisms were found in the cord.

J. H. HARVEY PIRIE.

ABSCESS OF FRONTAL LOBE OF TRAUMATIC ORIGIN. F. L. (422) TAYLOR, *N.Y. Med. Journ.*, May 1908.

THE clinical report of the case of a man who was struck with a brick above the right eye in May 1906. The man was not rendered unconscious by the blow, and returned to his work on the next day.

A month later an abscess developed round the wound. It was opened, some spicules of bone were removed, and the wound healed.

Thereafter, however, his intellect became sluggish. He could not sleep, and in addition severe occipital headache and frequent vomiting developed. When seen in September 1906 he was emaciated, yawned every few minutes, was apathetic, and had

a bilateral optic neuritis. He had no Bakinski, but there was incontinence of urine and fæces, and persistent vomiting.

An incision was made in the line of the old cicatrix, the dura was opened, a large hypodermic needle was pushed into the frontal lobe, and two ounces of pus were evacuated. As a result of the operation the man regained his intelligence, could control his urine and fæces, and stopped vomiting.

He remained well until October 1907, when the severe headache returned, and in addition he had convulsions which came on three days before, and occurred at ten-minute intervals for twenty-four hours before his death. The writer thinks that the return of these symptoms was due to the formation of a secondary abscess, and points out the importance of carefully watching cases that have been operated on with apparent success.

D. K. HENDERSON.

PALATO-LARYNGEAL HEMIPLEGIA. ROSE and LEMAÎTRE, *Ann. (423) de mal. de l'oreille, du larynx, du nez et du pharynx*, November, 1907, p. 467.

IN this paper the varieties, anatomical and pathological, of palato-laryngeal hemiplegia are discussed in a systematic and lucid manner. The actual anatomical innervation of the palate and larynx is still *sub judice*: the authors uphold Willis' contention that the bulbar roots of the spinal accessory should be considered as belonging to the vagus: in this sense the view that the palate and larynx receive practically all their nerve supply from the vagus is widely supported. The sensory supply of the palate is from the fifth: the laryngeal and pharyngeal afferent fibres belong to the tenth.

1. *Palato-laryngeal hemiplegia of peripheral origin.*—Such cases are rare. For a space of about two centimetres, immediately below the jugular foramen, a lesion of the vagospinal nerve will produce the form of paralysis under discussion: lower down, the pharyngeal branch leaves the vagus. Several instances are on record where the syndrome has resulted from disease in the neighbourhood, notably tuberculous glands and new growths.

2. *Of radicular origin.*—Basal lesions such as tumours or syphilitic pachymeningitis may occasion a palato-laryngeal hemiplegia. It is rare to find that there is a concomitant sensory paralysis.

3. *Of intrabulbar origin.*—Cases of palato-laryngeal hemiplegia of bulbar origin are not infrequently associated with a crossed anæsthesia, but no motor affection of face or limbs. It is supposed that a vascular lesion of the postero-inferior cerebellar artery, or

of the vertebral artery beyond the origin of the former, produces the syndrome. The association of the clinical phenomena with definite parts of the vagal nuclei is still a matter of difficulty. In most cases both nuclei and vagal fibres are involved. Paralysis of the palate certainly seems to depend on a lesion of the anterior part of the nucleus ambiguus. The occasional occurrence of certain (cervical) sympathetic symptoms in this syndrome is a point of considerable interest. In tabes, syringomyelia, and acute inferior polioencephalitis, palato-laryngeal hemiplegia may be encountered, as also in certain tumours of the area under consideration.

4. *Of supranuclear origin.*—A few rare cases have been described, notably those of Garel and of Déjérine. Paralysis of palate and vocal cord is known as the syndrome of Avellis; paralysis of palate, cord, sternomastoid and trapezius, the syndrome of Schmidt; paralysis of palate, cord, and tongue, the syndrome of Hughlings Jackson (incomplete); and of palate, cord, sternomastoid, trapezius, and tongue, the syndrome of Hughlings Jackson (complete).

S. A. K. WILSON.

HEMIPLEGIA WITH UNILATERAL OPTIC ATROPHY. (Clinical (424) Lecture.) R. T. WILLIAMSON, *Brit. Med. Journ.*, June 6, 1908.

IN this lecture four cases are described, in which there was atrophy of the *right* optic disc (causing loss of vision in the *right* eye) with hemiplegia of the *left* side.

These cases are examples of a peculiar combination of symptoms—optic atrophy on one side, with hemiplegia on the opposite side. The explanation suggested is that the symptoms were produced by obstruction (thrombosis) of the internal carotid and middle cerebral arteries on one side, with occlusion of the central artery of the retina (on the same side) by thrombosis (or embolism) spreading from the thrombus in the internal carotid. The pathological examination in two of the cases, and the ophthalmoscopic changes in one case, were in favour of this view. (A diagram illustrates the situation of the lesion.)

AUTHOR'S ABSTRACT.

DIAGNOSIS OF TUMOURS AND OTHER LESIONS IN THE (425) CEREBELLO-PONTILE ANGLE. T. H. WEISENBURG (Philadelphia), *Journ. Amer. Med. Assoc.*, April 18, 1908.

THE paper is concerned with the value of the different symptoms found in lesions of the cerebello-pontile angle, and five cases are reported, three with necropsy.

Two cases in which a tumour grew from the dura and covered

the petrous portion of the temporal and occipital bones gave symptoms of a lesion of the cerebello-pontile angle sufficient in one case to cause an operation to be done.

In the third patient there was almost entire absence of headache, nausea, vomiting, vertigo and choked disc, yet a cerebello-pontile angle lesion was diagnosed, and operation done and recovery followed.

The fourth patient after middle ear disease developed rigidity of the back and neck, increased reflexes, sluggish pupils, choked disc, cerebellar gait, inco-ordination of upper and lower limbs, and involvement of the left 6th, 7th and 8th nerves. Autopsy showed an abscess of the pia in the left cerebello-pontile angle.

The fifth patient showed occipital and temporal headache, gradual to almost complete loss of vision, double choked disc, paralysis of the entire 7th nerve, unsteadiness of gait and station, marked tremor, especially in the right upper limb, all the reflexes were increased, legs spastic; the autopsy showed a tumour filling the fourth ventricle and the posterior part of the aqueduct of Sylvius, a part growing outward into the left cerebello-pontile angle.

The paper furnishes much useful information concerning the interpretation of the clinical manifestations of cerebello-pontile lesions from carefully selected and well-studied cases.

C. H. HOLMES.

SYMPTOMS OF PONTINE TUMOURS. PARALYSIS OF ASSOCIATED EYE MOVEMENTS, AND LOSS OF CORNEAL REFLEXES. (Sur quelques symptômes des tumeurs de la protubérance. Les paralysies des mouvements associés des yeux et la perte des réflexes cornéens.) RAYMOND and CLAUDE, *L'Encéphale*, March 1908, p. 264.

THE symptoms of this case pointed to a tumour of the right half of the pons, with a crossed motor and sensory paresis. The patient was, in addition, unable to look either to right or left, and presented bilateral corneal anaesthesia, with loss of the corneal reflexes. The authors appear struck with the rarity of corneal anaesthesia when the rest of the trigeminal area is normal (yet this condition is not so very rare), and think it possible that certain trigeminal fibres run in the posterior longitudinal bundles which may be considered a path of motor and sensory fibres concerned in associated reflex movements of the eyelids and eyeballs. They remark, however: "Hâtons-nous de dire qu'il ne s'agit ici que d'une hypothèse qui ne s'appuie sur aucun fait physiologique ou anatomo-clinique." S. A. K. WILSON.

CEREBELLAR TUMOUR WITH PROPTOSIS. PARKINSON and
(427) HOSFORD, *Ophthalm. Rev.*, May 1908, p. 133.

THE patient during life presented somewhat anomalous symptoms, including great proptosis of each eye, equal on the two sides. There were unmistakable indications of intracranial neoplasm, and post-mortem a fibro-psammoma about the size of a pigeon's egg was found on the under surface of the right lobe of the cerebellum, pressing on the pons and medulla. The ventricles were much dilated, "and the cerebral cortex flattened and thinned, and tunics of nerve sheath distended."

S. A. K. WILSON.

PAPILLOMA OF THE CHOROID PLEXUS, Etc. (*Papillome des*
(428) *plexus choroides du IV^e ventricule, etc.*) VIGOUROUX, *Rev. neur.*, April 15, 1908, p. 281.

THE patient suffered for some years from an incessant escape of cerebrospinal fluid by the nose. He developed epileptiform seizures later, of a general nature, followed by mental confusion for some days. It was observed that the cerebrospinal discharge disappeared before the onset of fits, and reappeared thereafter. During the fits considerable bilateral exophthalmos was noted. At the autopsy the cavity of the fourth ventricle was found to be filled with a tumour resembling hypertrophied choroid plexus. The sella turcica and the pituitary fossa were abnormally large, and occupied by a cystic formation of the meninges, and in addition there were several apertures in the cribriform plate of the ethmoid, communicating with which were funnel-shaped prolongations of the dura, perforated at the distal ends. This was probably due to extreme intracranial tension, and not, as has been supposed, the result of a congenital malformation of the cribriform plate, with encephalocele.

S. A. K. WILSON.

LARGE ANEURISM OF THE SYLVIAN ARTERY (*Anévrysme*
(429) *volumineux de l'artère cérébrale ou sylvienne.*) A. SOUQUES,
Nouv. Icon. de la Salpêtrière, mars-avril 1908, p. 108.

THE symptoms first appeared when the patient, a man of 65, was 10 years old, in the shape of paroxysmal headaches, lasting a quarter of an hour or so, and vomiting. When 29 the headaches became more severe, localised to right side, and he had an attack of aphasia and left-sided hemiplegia. At 30 he became blind, the

hemiplegia gradually improved. When 63 years of age there was almost no trace of the hemiplegia, no objective sensory disturbances, reflexes normal save fan sign on left foot and a positive Oppenheim's sign. He was still having vertiginous attacks with headache and vomiting about once a fortnight, as he had had nearly all his life. There was optic atrophy following neuritis in both eyes, and no vision. Delusions of persecution now began to appear, and at 65 he committed suicide. Post-mortem there was found an aneurism of a branch of the right sylvian artery the size of a hen's egg. The other cerebral arteries showed a considerable degree of atheroma.

J. H. HARVEY PIRIE.

ON THE MECHANISM OF GLIOSIS IN ACQUIRED EPILEPSY.

(430) E. E. SOUTHARD, *Amer. Journ. Insan.*, No. 4, 1908.

THE writer terms his theory a micro-physical one, in that it seeks a logical basis for the epileptic discharge in certain intimate alterations of pressure in the central nervous tissues. He is inclined to think that by combining the consideration of gliosis on the one hand and of certain stratigraphic changes on the other, a structural basis can be laid for the understanding of the inhibitory mechanism which underlies epilepsy.

The theory expounded lays claim to originality in two directions—(1) In setting forth the properties of a typical epileptogenic focus in the cortex; (2) the nature of that change in cortical tissue which favours epileptic discharges.

The characteristic features of a typical prime focus are described as the separation of a normal cell group from its normal control by other cell groups, and the impact upon the receptive surfaces of these normal cells of a steady, intimate, abnormal pressure—both segregation and pressure effected by neuroglia overgrowth.

That feature of cortical tissue which favours the spread of epileptic discharges is described as due to a simplification of cell arrangements, arising in the destruction of controlling elements with maintenance of motor elements.

The peculiar features of the epileptic discharge depend upon the inertia of currents travelling in simplified areas, and upon the lack of energy-absorbents *en route*. The cerebral areas normally escape automatism through a multitude of synaptic connections; under epileptic conditions the cerebral mechanism approaches in fatality the spinal mechanism.

Under this conception, epilepsy and phenomena like clonus are readily perceived to belong to a single logical group.

The paper is illustrated by some very good photo-micrographs of the brain-cortex, showing the distribution of the glial changes

affecting chiefly the small cells of the second layer and the cells of the stratum granulosum.

JOHN TURNER.

EPILEPSY—THE SO-CALLED IDIOPATHIC FORM. TUCKER,
(431) *N.Y. Med. Journ.*, June 6, 1908.

THIS paper contains merely an account of the clinical forms, diagnosis, pathology, and treatment of epilepsy, largely based on the views of W. Aldren Turner. The author expresses himself as in the main in agreement with the views concerning the pathology of the disease as expressed in Turner's book on epilepsy.

JOHN TURNER.

A REPORT OF TWENTY-SEVEN CASES OF CHRONIC PROGRESSIVE CHOREA. A. S. HAMILTON, *Am. Journ. of Insan.*,
(432) Vol. lxiv., No. 3, Jan. 1908.

THE writer gives full clinical reports of these twenty-seven cases, seventeen of which he examined personally, and ten of which are taken from the records of the institution at Independence, Iowa. He maintains that there is no essential difference between hereditary chorea and senile chorea, as seventeen of these cases which were originally diagnosed as senile chorea were found later, on a more thorough investigation being made, to have a well-marked hereditary predisposition. In the writer's cases there is a history of the condition in the immediate relations in twenty-four instances. In the remaining three the history is lacking, and it is pointed out that the ordinary nervous and mental diseases are conspicuous by their absence in these patients. Rheumatism was present in but five cases, and certainly plays no such important rôle here as in the chorea of childhood.

Manifestations of ordinary physical disorders were not more frequent in most parts of the body than would be expected in a group of people, many of whom were well advanced in life. It is noteworthy that in some of the patients in whom the movements were violent and continued, not only during the day, but also through a considerable portion of the night, there was no complaint of fatigue. The part of the body first affected varied considerably; sometimes the movements were first noticed in the hands, sometimes in the lower extremities, and sometimes in the face. Ultimately, however, the whole body is involved in most instances, though the movements may be more severe in some parts than in others. Speech defect appeared in most of the cases, though at times only when the disease was far advanced.

In all the writer's cases the movements were absent during sound sleep, but not always during light slumber.

A well-marked increase in muscular tonicity was present in nearly all of the well-developed cases, and in several it was a very striking phenomenon.

In all the cases recorded here there was mental impairment, the condition being a gradually increasing dementia, marked irritability, and often distinct delusions of persecution.

D. K. HENDERSON.

SOME OF THE MOTOR PHENOMENA OF CHOREA CLINICALLY CONSIDERED. F. R. FRY, *Journ. Amer. Med. Assoc.*, Vol. i., No. 18, May 1908.

THE writer distinguishes between tic and choreic movements. Tic is held to be an affair of a higher cerebral level and closely related to psychic functions. The choreic movement, on the other hand, is due to some defect in the motor inhibitory apparatus. Tic is evidence of a more pronounced neuropathic predisposition.

Many of the subjects of chorea are very silent, and the difficulty in these cases is mostly one of motility, but in other cases it is due to a sluggish general mentality.

D. K. HENDERSON.

DISTURBANCES OF SENSATION OF CEREBRAL ORIGIN AND SPINAL TYPE. (Zur Frage der zerebralen Sensibilitätsstörungen von spinalem Typus.) STRÄUSSLER, *Monatsschr. f. Psych. u. Neurol.*, May 1908, p. 381.

IN a case of Jacksonian epilepsy affecting the right arm and face followed by paresis in the same distribution, the author found certain disturbances of sensation approximating to a spinal type. Thus with a slight subjective numbness of the whole of the right half of the body was coupled an objective loss to pain, touch, and temperature stimuli in an area corresponding more or less exactly to parts of the fourth, fifth, and eighth cervical segments, and of the first to the fourth dorsal segments. Passive movements of the fingers (and toes) on the right side were not recognised at all. Stimuli were incorrectly localised in the forearm, hand and fingers. The right hand was astereognostic. At a later stage improvement took place. The patient was frequently wrong in his localisation in his fingers, although he recognised the nature of the stimulus: it was almost constantly referred to the middle finger. Passive movements were usually recognised, but referred

to the wrong finger. The error of atopognosis was chiefly a proximal one.

In a case such as this, of considerable scientific value, it is to be regretted that the examination was not more thorough. There is no reference to the exact nature of the stimuli used for testing topognosis, nor to the degree of the proximal or other errors that were noted. There is no record of the nature of the passive movements employed, and whether any distinction was appreciated by the patient between flexor and extensor movements. The desirability of a careful discrimination between the protopathic and epicritic types of sensation does not appear to have been realised, as there is no reference whatever to their diagnostic value as regards the localisation of the lesion. The term "motor atopognosis" for errors in the recognition of passive movements is ambiguous. It would have been interesting to know whether the patient exhibited astereognosis in the exact sense of the word, as the description suggests his condition may really have been one of tactile agnosia.

S. A. K. WILSON.

TOTAL ANÆSTHESIA. (*Totalanæsthesie.*) L. E. BREGMAN, (435) *Neurol. Centralbl.*, Juni 1, 1908, p. 498.

TOTAL anæsthesia is one of the rare sensory symptoms of hysteria. The symptom is of great interest in connection with (1) the influence of sensory impressions upon the accomplishment of movements, and (2) the importance of sensory impressions for consciousness. Strümpell has recorded a case in which there was complete loss of sensation, excepting that sight in one eye and hearing in the opposite ear were retained. This patient went off into a deep sleep whenever the eye and ear were covered. Strümpell deduced from this case that afferent impressions are necessary to maintain the waking state. This explanation does not, however, account for the periodicity of sleep, or for the fact that we go to sleep in spite of the external impressions which are constantly reaching our cerebra. Strümpell's observation has been confirmed by Heyne and others. Pronier has recorded a negative observation, and the case here reported is also negative.

The patient, a girl aged 26, had suffered for twelve years from attacks in which she stated that she lost all feeling in the limbs and body as well as taste and smell. She felt, she said, like a piece of wood in these attacks. Upon examination, there was found to be some general weakness, although she was able to walk and run quite well. There was total anæsthesia for touch, pain, and temperature over the whole body. The muscle sense, sense of position, sense of movement, and pressure sense were all lost, as was the perception of electrical currents. Taste and smell were

lost. There was full vision in both eyes, although the fields were somewhat contracted. Hearing was also preserved. The patient was able to stand and walk quite well when the eyes were closed. All movements were carried out quite well without a trace of ataxia. When the eyes were bandaged and the ears stopped the patient lay for two hours without manifesting any tendency to go to sleep. The sleep described by Strümpell is thus no necessary accompaniment of total anæsthesia. Strümpell, who interpreted his experiment with all reserve, admitted later that the sleep induced in his patient resembled that of hypnosis. An attempt to hypnotise Bregman's patient failed.

EDWIN BRAMWELL.

INTEGRITY OF STEREOGNOSTIC FUNCTION AND ALL FORMS (436) OF SENSATION IN A CASE WITH A LESION OF THE LEFT PARIETAL LOBE. A. GORDON (Philadelphia), *Med. Rec.*, April 18, 1908.

A MAN of 58 shot himself in the right temple, and the bullet lodged in the left parietal region; he had no recollection of the shooting and remembered little for several months afterward. His reflexes, motor power and sensations were normal and equal on both sides; he could understand written or spoken words and he could write; the eye examination was practically negative.

After the location of the bullet by X-ray examination, and both before and after operation, the author made numerous examinations for sensation, and invariably the sense of touch, pain, temperature and stereognostic sense were found to be normal and equal.

The operation was performed on the left parietal lobe, the bullet found and removed about one half inch from the cortex. The patient recovered, and there were no more epileptiform seizures.

C. H. HOLMES.

BONE SENSATION. (*La sensibilité osseuse.*) EGGER, *Rev. neur.*, (437) April 30, 1908, p. 345.

In this communication Egger restates his views on the sensation of vibration, and criticises the arguments that have been variously advanced against his theory. It cannot be said that his criticism invariably carries conviction.

S. A. K. WILSON.

STEREOGNOSIS AND SYMBOLY IN THE LOWER EXTREMITIES.

(438) (*La Sensibilité stéréognostique et la symbolie aux membres inférieurs.*) MARBÉ, *Rev. neur.*, April 30, 1908, p. 351.

It is highly desirable to preface any communication on "stereognosis" and "symboly" with a precise definition of these controversial expressions, as understood by the author. In the present instance this has not been done; one is accordingly at a loss to know what he intends by them; the impression conveyed is that they are either identical or interchangeable, since in one passage the author says, "l'astérognoisie coïncide avec l'ataxie," and immediately below he repeats, "cette coexistence de l'asymbolie et de l'ataxie," etc. No good purpose can be served by such confusion. All of the cases examined (the objects were applied to the patients' feet and toes) had some or other defect of cutaneous or deep sensation—rendering the interpretation of the author's results very ambiguous.

S. A. K. WILSON.

ON THE MECHANISM OF BABINSKI'S SIGN, OR THE PHENO-

(439) **MENON OF THE TOES.** (*Sur le mécanisme du signe de Babinski, ou le phénomène des orteils.*) NOICA, *Journ. de Neurol.*, March 1908.

THE relation of the toe phenomenon to a lesion in the pyramidal system of fibres has been established, but its precise nature and mechanism have given rise to considerable discussion. Babinski regarded it as a transformation of the normal plantar reflex, while Van Gehuchten saw in it two distinct phenomena—abolition of the normal plantar reflex and the production of a new reflex (extension of great toe).

These views have been disproved, for it is a clinical fact (Crocq, Marinesco, Noica) that in some spastic paraplegiacs both reflexes exist: on exciting the external border of the foot, Babinski's phenomenon is produced; on exciting the internal border, the plantar reflex (flexion) results. Marinesco has shown that if the external and internal borders of the foot are simultaneously excited, it is the flexion-reflex that prevails, or else there is a sort of hesitating action of the great toe, which takes up a position midway between flexion and extension. Sometimes it is the extension-reflex which predominates. Both Crocq and Marinesco hold that in this case there is a struggle between the flexor and extensor muscles for the production of the reflex, and the victory most often falls to the flexors. Even in those cases where only the extension reflex is obtained, it does not follow

that the flexor-reflex is abolished, but simply that the contraction of the extensors prevails over that of the flexors.

Dr Noica admits the facts, but rejects the explanation above given. He contends that in spastic conditions the flexor muscles are the stronger, and if the character of the reflex depended upon a contest between the two sets of muscles, then the flexion-reflex should be constant in spasmodic states. He reminds us that a condition of muscular repose is essential for the production of a reflex, and argues that in strongly spasmodic states we may fail to obtain a plantar reflex, not because it is abolished, nor yet because it is inhibited from manifesting itself owing to the presence of Babinski's reflex, but for the simple reason that the degree of contracture prevents it showing itself. The less the contracture, the more likely are we to obtain both reflexes, and it is in these cases that simultaneous excitation of both borders of the foot may be followed by a struggle between flexors and extensors resulting in a flexor-reflex. If the contracture becomes still less marked, we may get a well-marked plantar reflex and only the outline of the Babinski reflex. If the hemiplegia is cured, the plantar reflex is exaggerated, and excitation of either the internal or external borders of the foot will always be followed by the plantar flexor-reflex.

The toe phenomenon is not, strictly speaking, an abnormal reflex. It is normal in the newly born, and in young infants for a variable length of time.

The flexor plantar reflex only makes its appearance, says Dr Noica, with the development of the function of walking, and thereafter supersedes the extensor reflex, which diminishes and finally disappears.

In the young infant the flexor muscles of the limb are the stronger. As a rule, the foot is bent dorsally on the leg, the leg flexed on the thigh, and the latter on the pelvis. When walking is required, the extensor muscles of the foot become more developed, and so the normal man has more power to lower the point of the foot and to flex the toes downwards than to bend the foot and toes dorsally on the leg. In the adult, excitation of the sole of the foot, especially the internal border, is consequently followed by a downward flexion of the toes (classic plantar reflex), for the motor centres of the muscles concerned are now much more active than the centres co-ordinating the opposing group of muscles (dorsal flexion of toes or extension reflex).

Noica brings forward strong evidence in support of his argument, and concludes "that with the development of walking, which is in direct relation with the development of the pyramidal tract, the reflex of Babinski disappears or is reduced to a shadow, and that if the function of the pyramidal tract is interfered with,

this reflex re-appears, and, further, persists with the character described by Babinski, so long as the function of the pyramidal bundle is not re-established."

J. H. MACDONALD.

THE SIGNIFICANCE OF THE TENDO ACHILLIS JERK. (Ueber (440) *die Bedeutung des Achillessehnenreflexes.*) CONZEN, *Münch. Med. Woch.*, 1908, No. 19, p. 1014.

CONZEN investigated the tendo Achillis jerk in 3290 cases in F. H. Hoffmann's clinique at Leipzig. He found that normally it was always present, and that it was only absent or diminished in pathological conditions of the nervous or muscular systems. Babinski's method of examination was adopted, which consists in making the patient kneel on a chair with the feet hanging free. In some cases of varicose veins the ankle jerks were diminished or absent, an occurrence which Conzen attributes to slowly developing neuritic changes. In one of two cases of alcoholic neuritis in which the knee jerks were present, both ankle jerks were completely absent, and in the other the right ankle jerk was absent, while the left was very feeble. In cases of sciatic neuritis diminution or abolition was sometimes preceded by a slight exaggeration of the knee jerk. (A similar observation was made by the abstractor in examination of the tendo Achillis jerk in diphtheria. *V. Brain*, 1905, p. 71.) In a case of progressive muscular atrophy both ankle jerks were absent, while the knee jerk was present on the left. In tabes Conzen found that the ankle jerk was lost much earlier than the knee jerk.

These observations show that examination of the tendo Achillis jerk should never be neglected, since it is as constant in health and as liable to be affected in nervous disease as the knee jerk, while even unilateral abolition is pathological.

J. D. ROLLESTON.

HYSTERICAL MUTISM. (Ein Beitrag zur Geschichte des hysterischen Mutism.) FRANZ JAHNEL, *Neurol. Centralbl.*, Juni 1, 1908, p. 512.

IN an old book entitled "Magazin des Ausserordentlichen in der Natur, der Kunst und im Menschenleben," published in 1815, the author has met with an account of an interesting case of hysterical mutism occurring in a man who lived so long ago as 1653. The patient when ten years of age was nearly drowned, and soon after lost his speech for gradually increasing periods each day. Until his death nearly fifty years later he only spoke between the hours

of twelve and one each day. Even when no clock was near he knew to a minute the hours of twelve and one. Only on two occasions did he break this rule during his life, and on each of these occasions he was suffering from fever. For two or three days before his death, however, he was able to speak quite well. Mendel has recorded a case in which the patient for a year was only able to speak between the hours of six and nine.

EDWIN BRAMWELL.

ON ALLEGED WORD-DEAFNESS IN MOTOR APHASIA. (Über (442) die angebliche Worttaubheit der Motorisch-Aphasischen.)
LIEPMANN, *Neurol. Centralbl.*, April 1, 1908, p. 290.

PIERRE MARIE has maintained that all cases of Broca's aphasia exhibit a greater or less degree of imperfection in understanding speech. A motor aphasic is thus a sensory aphasic with anarthria. The fact that a motor aphasic is frequently unable to perform the somewhat complicated tests which that author employs is far from convincing, however, inasmuch as they do not really test his ability to understand written or spoken language, but rather his capacity for retaining recent impressions.

Apart from this, it is undoubtedly the case that with motor aphasia a degree of word-deafness may occur; but the conclusion that this word-deafness is part of the clinical picture of motor aphasia is unwarrantable, for the following reasons:—

1. Many patients who have been diagnosed *intra vitam* as cases of motor aphasia are found subsequently to have had lesions which are far from being limited to Broca's area (understanding by that term the posterior end of the third left frontal convolution, the operculum, and the anterior part of the insula). That such cases are called motor aphasics is due to the frequently overlooked fact that their word-deafness is so often transitory. As long ago as 1874 Wernicke emphasised the transitory nature of word-deafness in many left hemisphere lesions. Either the right temporal lobe, or the rest of the left temporal lobe, assumes the function that has been for a time lost. It is a common experience in cases of sensory aphasia to find the word-deafness diminishing, while the paraphasia and reading and writing defects remain. In discussing the question of motor aphasia those cases only must be considered where the lesion is *strictly* limited to Broca's area.

2. A diagnosis of motor aphasia is frequently made erroneously: as when a patient understands fairly well, but is of very limited speech because of paraphasia. When a lesion in the temporal lobe is subsequently discovered, the physician is astonished at the insufficiency of the classical theory instead of at his own ignorance.

Sensory aphasia and complete word-deafness are not identical, nor are motor aphasia and difficulty in the expression of speech. The motor aphasic is entirely, or almost entirely, word-dumb. The difference between the motor and the sensory aphasic is *not* that the former cannot speak properly, while the latter cannot understand properly. The usual lesion in a case of imperfect (however slight the imperfection) comprehension with considerable defect of expression is in the temporo-sphenoidal lobe.

3. Not all word-dumb patients are cases of motor aphasia, *pace* Pierre Marie. It is years since the question was first discussed whether patients with complete sensory aphasia may not therefore be speechless. Bastian and others have laboured to make it clear that in the majority of mankind speech depends on the revival of the sounds of words, and that the functional activity of Broca's centre depends on the functional integrity of Wernicke's. Complete speechlessness is one of the elements of sensory aphasia. It is true that this loss of speech is rarely if ever found in unilateral (left-sided) lesions of the temporal lobe. In bilateral lesions, however, the patient becomes absolutely word-dumb (Mott, Liepmann, etc.), although he is not suffering from Broca's aphasia. Transcortical motor aphasia of this kind is as different from Broca's aphasia as is hysterical mutism or bulbar anarthria. In motor aphasia of frontal origin there is never word-deafness. A case of word-deafness and word-dumbness of bilateral temporal origin can be distinguished from a case of Wernicke's plus Broca's aphasia by the circumstance that in the latter the power of understanding speech may return to some extent, as well as often by the clinical history of the case.

4. Many motor aphasics are dyspraxic, and thus are not uncommonly supposed to be suffering from word-deafness.

While it may be admitted that some cases of motor aphasia reveal a "defect of intelligence," this defect is always of the nature of a failure to understand the meaning of words, whereas the typical cortical sensory aphasic's failure is in understanding the sound of words. In the motor aphasic's case this "defect of intelligence," if present, indicates that he cannot associate quickly enough the words of a sentence with their corresponding inner meaning. If the two are not associated at once, the patient may appear not to understand. Such a condition may readily arise in any normal individual, without his ever being considered word-deaf. It may very well be that such impairment is the result of a general cerebral change (of an arterio-sclerotic, atrophic, or chemical kind) apart from the actual local lesion.

S. A. K. WILSON.

THE CONDUCTION APHASIAS. MINGAZZINI, *L'Encéphale*, Jan. (443) 1908, p. 1.

ACCORDING to Wernicke and Lichtheim, a lesion situated so as to interrupt the fibres which pass from the auditory speech (word) centre to Broca's area, *via* the insula, will produce an aphasia of conduction, and this conduction aphasia is characterised by one special symptom, *viz.*, paraphasia. This paraphasia must not be confused with that which accompanies word-deafness or word-blindness, still less with that which accompanies motor aphasia. In conduction aphasia the auditory word-image, as a result of the interruption of the conduction path, cannot exercise its influence on the choice of motor word-images. The patient thus afflicted understands his paraphasias and seeks in vain to remedy them. In addition, he always reveals some defect both in reading and in writing.

Proof of this theory, however, is still to seek. Various paraphasic cases have been found, it is true, to present a lesion of the insula, but there has always been an additional involvement of regions outside the insula itself. If the posterior part of the insula be destroyed, one finds clinically an incomplete auditory aphasia, as well as paraphasia. If the anterior portion of the insula be destroyed, we have a motor aphasia (Bastian, Déjérine) identical with what is known as Broca's aphasia. Yet this statement, too, requires reconsideration. The author adduces pathological evidence to show that the auditory and motor word-centres have a wider extent than is usually supposed, and that it may be impossible to define their limits precisely. He gives the details of a case of partial lesion of Broca's convolution on both sides, when the patient was unable to put in order the syllables forming the words which he wished to pronounce, the result being a paraphasia strictly comparable to what is found with a lesion of Wernicke's area. Broca's lobule registers glosso-kinæsthetic memory images of syllables, not of words, and it arranges them in proper order on stimuli from the auditory word-centre; thus while clinically a paraphasia from a Broca lesion and a paraphasia from a Wernicke lesion are identical, the mechanism of their production is different in the two cases: in the latter the stimulus is wanting, the glosso-kinæsthetic images of letters and syllables remaining intact; in the former the stimulating element is present, but it no longer finds the motor-images which it should co-ordinate.

There are cases of lesion of Wernicke's zone and the left lenticular nucleus in which, in spite of the preservation of the third left frontal convolution, the patient suffers not merely from auditory aphasia but also from almost complete motor aphasia,

and on these cases Pierre Marie has laid stress in his revision of the question of aphasia. The explanation is that the fibres which descend from Broca's lobule carrying the impulses from that area pass by the antero-lateral part of the putamen. If the posterior part of the putamen be injured, dysarthria results, according to Mingazzini. The question of the distinction between dysarthria or anarthria and aphasia is difficult. The author believes that lesions of the fibres from Broca's centre below the level of the putamen produce dysarthria, above that level aphasia. He supposes that a new order of fibres convey from the putamen and the pontine nuclei motor influences corresponding to the word or syllable images transmitted from Broca's convolution. This bundle of fibres is intimately associated with the ordinary cortico-bulbar path for the innervation of facial and other musculatures and the pontine nuclei; hence the frequent association of their paresis with dysarthria.

S. A. K. WILSON.

A CONTRIBUTION TO THE STUDY OF APRAXIA. S. A. K. (444) WILSON, *Brain*, 1908, p. 164.

THIS paper contains a historical retrospect over the field of agnosia, asymboly, and apraxia, and in view of the ready confusion which an inaccurate use of these terms occasions, their meaning is carefully determined. Due recognition is made of the contributions of Dr Hughlings Jackson to the subject, now more than forty years ago, contributions of which all previous writers on apraxia have apparently been unaware. Numerous original clinical observations of apraxia are reported. Attention is drawn to the occurrence of apraxia in chorea.

Apraxia may briefly be defined as "inability to perform certain subjectively purposive movements or movement-complexes, with conservation of motility, of sensation, and of co-ordination." There are various kinds of impairment of cerebral function, producing defect of movement: the position occupied by apraxia may be thus indicated:—

1. *Cortical blindness, cortical deafness, cortical sensory paralysis*: loss of visual, auditory, and cutaneous ingoing sensory impressions.
2. *Cerebral ataxia*: loss of afferent kinæsthetic impressions, resulting in erroneous estimation of range, power, etc., of movement.
3. *Mind-palsy (Seelenlähmung)*: incapacity for movement from loss of kinæsthetic images and memories for complex movements.
4. *Agnosia, mind blindness, mind deafness, etc.*: conservation

of sensation, but failure of recognition; loss of sensory memory pictures.

5. *Ideational agnosia*: loss of the spatial associations and inter-connections that build up the idea of an object from its component ideas.
6. *Ideational apraxia*: defective synthesis of the ideational components of a movement-complex: defective psychical "sketch" of movements or acts.
7. *Motor apraxia*: intactness of the cortico-muscular apparatus, but inability to translate a normally produced idea of a movement into the corresponding movement-form.
8. *Motor paresis or paralysis*.

It is advisable to restrict the term apraxia to motor apraxia. Sensory apraxia is misleading: the proposal is made to describe apraxic phenomena due to agnosia as "agnostic apraxia," or apraxia secondary to agnosia. That apraxia is often thus determined is of course obvious. Various instances are recorded and analysed. Apraxia may be caused by perseveration. Inattention, incapacity for retaining recent impressions, and absence of initiative are also factors in the production of apraxia.

The relation of apraxia to aphasia is alluded to, but it forms too important a subject to be handled in the limits of one paper. The pathological anatomy of apraxia is vastly interesting and instructive. Cases are quoted to show the importance of lesions of the corpus callosum, of the left frontal lobe, and of the left senso-motorium in the determination of apraxia. The article finishes with a scheme for the examination of apraxic patients. The subject of the paper does not admit of a satisfactory résumé.

AUTHOR'S ABSTRACT.

A CASE OF WORD-BLINDNESS WITH AGRAPHIA, IN A LEFT-(445) HANDED HEMIPLEGIC. (Su d'un caso di cecità verbale con agrafia in una mancina 'emiplegica.) G. D'ABUNDO, *Riv. Ital. di Neuropat., Psichiat., ed Elettrotet.*, 1908, p. 257.

It has recently been argued that, inasmuch as speech is a purely human function, nothing is to be learnt about it from experimental pathology. In view of this argument we shall need to consider a fresh series of clinical observations. Marie's revolutionary views on this subject also necessitate renewed research into the problem of the localisation of the neuropsychical factors of speech.

The case described is that of a woman, 31 years of age. She was left-handed as a girl, though she could, if need be, both write and sew with her right hand. Her school education was very

scanty. At the age of 15, two days after receiving a great fright, she became unconscious, and, on coming to herself, was found to be completely paralysed on the left side, as well as being word-blind; subsequently agraphia also developed.

A year later, having to some extent recovered the use of her limbs, she was seized, while carrying a heavy weight, with a left-sided convulsive attack; from that time onwards any attempt at voluntary movement of the left arm threw it into a spasmodic choreiform condition. The alexia and agraphia remained permanent.

On examination, some years later, patient's general health was found to be good. Besides the features already described, there was diminution of tactile sensibility in the limbs of the left side, and marked left homonymous hemianopsia in both eyes. She was also frequently the subject of left-sided convulsive seizures, some of which consisted merely of flashes of light on the hemiopic side, others involving the left facial nerve, and others being typical Jacksonian attacks. She was not only unable to read a word, but could not even recognise the letters of the alphabet. Agraphia accompanied the word-blindness. Her intelligence and memory were good.

The leading symptoms of this case—hemiplegia, with hemihypæsthesia, hemiopia, and word-blindness—point to a lesion of the inferior and ascending parietal convolutions on the right side; patient being left-handed, the word-blindness would depend on involvement of the right angular gyrus.

The centre for the memory of movements necessary in writing would in this case be naturally developed by education in the left hemisphere. The noteworthy point is that the word-blindness caused by a lesion of the *right* parietal cortex had associated with it agraphia, a disturbance of the *left* hemisphere; for this there must be a psychical explanation.

This lesion involving the centre for visual word-memories was entirely unconnected with any intellectual deficiency; thus the case tends to maintain the distinction between mind-blindness and word-blindness.

Attempts at the re-education of this patient were much impeded by the hemiopia.

ARTHUR J. BROCK.

ON CONGENITAL WORD-BLINDNESS. (Ueber kongenitale Wortblindheit.) PETERS, *Muench. Med. Wchnschr.*, May 26, 1908.

THE writer gives a brief résumé of various publications on this subject since the pioneer observations of Kerr and Morgan in 1896, and states that no thoroughly typical case has yet been

recorded in Germany ; he then quotes two cases which have come under his own observation. The first was that of a boy of twelve, in whom the power of recognising written or printed words was badly developed, although in other respects he was quite normal. The second case was also in a boy, in whom, however, mental deficiency was superadded, the investigation of the case being thereby much complicated.

It is very noteworthy that almost all cases of congenital word-blindness have so far been published either by English or by English-speaking investigators ; this is probably because "the words of the English language, owing to their peculiar pronunciation (*eigenartige Aussprache*), are more difficult to read than words from other languages, which can be more easily learnt phonetically." Stephenson reports a case in which Latin words were read more easily than English.

In London the school medical officers estimate the occurrence of this disturbance as 1 case in every 2000 school children. The condition is far more frequent in boys than in girls.

As regards its pathology, the writer agrees with Morgan that it is due, not to degeneration, but to faulty development—to a congenital aplasia of the cortex in the region of the angular gyrus.

In regard to treatment, much may be done outside the schools by systematic training, and the writer considers this a profitable field for charitably-disposed ladies.

ARTHUR J. BROCK.

DYSPRAXIA WITH LEFT-SIDED HEMIPLEGIA. (*Dyspraxie bei (447) linksseitiger Hemiplegie.*) HILDEBRANDT, *Neur. Centralbl.*, June 16, 1908, p. 576.

THIS is a record of a case in which there was pronounced hemiplegia of the left side, with decided dyspraxia of the right hand. When young the patient had been left-handed, but on account of her being teased by her brother for this she had endeavoured to get over it and use her right hand. She had largely succeeded in correcting her early habit, but not entirely. Cases of left-handed dyspraxia, with right-sided hemiplegia, are not unknown, but so far as the author has ascertained few of the contrary have been recorded. Hence his communication of the above case.

JAS. MIDDLEMASS.

PSYCHIATRY.

ALCOHOL AS AN ETIOLOGICAL FACTOR IN MENTAL DISEASE. (448) HENRY COTTON, *Amer. Journ. Insan.*, April 1908.

THAT "alcohol accounts for an alarming number of commitments in the insane hospitals to-day, directly, indirectly, and through

inherited influence, and that the percentage is slowly but steadily increasing," is the conclusion the author of this paper draws from a statistical study of the question. An examination of the statistics of modern mental hospitals easily convinces one that alcohol is the direct cause of a large percentage of the cases of insanity treated in them. But that is not all. There are a vast number of habitual drinkers who, though not legally insane, are a danger to the community. Various illustrations are given of the evil effects of alcohol. Kraepelin's observations are of special interest, and tend to refute the idea that alcohol (in the form of beer-drinking) does not affect the German nation.

The indirect results of alcohol are equally important. The percentage of cases of mental and other nervous diseases in the production of which alcoholism may be considered as acting as a contributing cause is remarkable, *e.g.* in 46.6 per cent. of general paralytics alcoholic excesses were present. Kraepelin believes that one-third of the cases of general paralysis would be avoided if syphilitic subjects abstained from alcohol. It must further be borne in mind that three-fourths of the injections of syphilis occur during intoxication.

As to the inherited effects of alcohol, apart from the vexed question of the inheritance of acquired characters, there is not a little to support the view that alcoholism in the parents, especially at the time of conception, can exert a toxic influence on germ plasma. The percentage occurrence of miscarriage and abortion is greater in alcoholic than in non-alcoholic families. The effect of alcoholic parentage in idiots and epileptics some observers give as high as 85 per cent.

A. HILL BUCHAN.

ON A CLASS OF CHRONIC NON-MORAL ALCOHOLICS. (D'une (449) classe d'alcooliques chroniques amoraux. Envisagée au point de vue de la responsabilité légale.) SOUTZO fils and P. DIMITRESCO, *Ann. Méd.-Psychol.*, May-June 1908.

IN this article the authors deal with a class of chronic alcoholics whose moral and intellectual faculties have been impaired by their habits. The symptoms are described and cases given in illustration. The relation of such persons to criminal law—whether they can be considered responsible for their actions or ought rather to be regarded as subjects of a special mental disease requiring other than ordinary prison treatment—is discussed.

A. HILL BUCHAN.

EPILEPSY AND CHRONIC DELUSIONAL INSANITY—CONTRIBUTION TO THE STUDY OF COMBINED PSYCHOSES.

(*Épilepsie et Délire Chronique—Contribution à l'Étude des Psychoses Combinées.*) FR. MEENS, *Ann. Méd. Psych.*, May-June 1908, p. 353.

THIS paper is concerned with the possibility of the co-existence in the same individual of two distinct psychoses, a question already discussed by many eminent alienists, but about which unanimity of opinion does not yet prevail. Dr Meens records two cases which, in his view, conclusively prove that such a co-existence is possible. Theoretically speaking there appears to be no insuperable argument against its occurrence. In the case of most other organs it is quite well recognised that two quite different diseases may co-exist, each showing the main features which distinguish it when existing alone. There is, therefore, no *a priori* reason why the same condition may not be exhibited in the diseases of the brain which give rise to psychoses.

The first of the two cases recorded is that of a woman, who, at the age of eight, developed epilepsy, at the age of forty-one began to show symptoms of paranoia, and at the time of writing was aged sixty-six. For twenty-five years the two conditions co-existed, developing each along the lines usually followed by it, and not apparently influencing each other's progress to any marked degree.

The second case is that of a man aged fifty-six, who, since he was thirty-five, has manifested the symptoms of both epilepsy and chronic delusional insanity. It could not be ascertained whether the epilepsy was the first to make its appearance or not, and, if so, how long before. In his case also the two diseases have run their course side by side.

In discussing the question of combined psychoses the author rightly insists that care must be taken not to confound a distinct psychosis with accidental and frequently transient symptoms, such as depression, elevation, or confusion. These are symptoms common to many psychoses, and do not therefore by themselves indicate the existence of a second disease. By those who uphold the existence of combined psychoses various explanations of their contention are given. It is supposed that one is transmitted from the father, the other from the mother (Magnan). In the first case described by Dr Meens this was certainly not so. In the other the history was defective. By some it is suggested that the paranoiac delusions are the outcome of incorrect deductions made during the confusional state associated with the epileptic fit (Ziehen). But here again the author's observation is against this suggestion. Others, again, hold the view that epilepsy, by weaken-

ing the mental faculties, lays the affected individual open to attack by delusional ideas, which in a healthy mind would be resisted (Buchholz). This view is also set aside by the author as not warranted by the facts of his cases, as well as on other grounds. He considers the co-existence of two psychoses in one individual to be only an accidental coincidence which, in the absence of definite pathological knowledge, cannot yet be satisfactorily explained.

JAS. MIDDLEMASS.

THE DEVELOPMENT OF THE MODERN CARE AND TREATMENT OF THE INSANE, AS ILLUSTRATED BY THE STATE HOSPITAL SYSTEM OF NEW YORK. CARLOS F. MACDONALD, *Amer. Journ. Insan.*, Vol. lxiv., No. 4, p. 647.

THE author records what has been done in recent years to improve the unhappy lot of the insane in the State of New York.

As a result of a reform agitation by some philanthropic people, there was appointed in 1889 a State Commission in Lunacy, in which was vested plenary power in respect to the insane and the management of institutions for the insane, both public and private.

As an outcome of the investigations and recommendations of the Commission, the State Care Act was passed in 1890. By this means the dependent insane were looked after as wards of the State, and were treated and cared for in a humane way. Asylums, or State Hospitals as they are called, were built. Old institutions were put into a thoroughly sanitary condition, and overcrowding of patients was abolished. It was made compulsory for all improvements and reconstructions of a hospital and for all expenditure to be approved by the Commission. At the same time there was established a Pathological Institute, which was to be maintained for the benefit of all hospitals, and where scientific research would be carried out in the endeavour to elucidate the etiology and pathology of mental diseases.

Prior to the passing of the Act it was customary to transfer unrecovered patients, often at the end of a year, from asylums to poorhouses, where existed a deplorable state of affairs as regards management and treatment. This is now illegal.

The author points out many other important improvements which have accrued to the institutions for the insane, and some of the benefits derived by the patients as a result of this legislation.

The interests of the patients are safeguarded in a manner very similar to that in this country, and proper medical treatment is now administered.

Certification of an insane person, in order to commit him to a hospital, can be done only by a qualified examiner, whose qualifications must be certified to by a Judge of a Court of Record, and

the certificate must be filed in the office of the Lunacy Commission. Resident officers in State Hospitals are required to pass a competitive civil service examination.

A most praiseworthy step has been taken in the formation of the State Charities' Aid Association, the object of which is to render temporary assistance and friendly aid and counsel to needy persons after their discharge from the hospital. It was believed that timely help and encouragement to such persons would serve to prevent relapse in many cases, and the results thus far reported justify the opinion that the belief was well founded.

While the mentally afflicted have benefited in every way under the "Insanity Law," a great saving in the maintenance of the hospitals has been effected.

R. DODS BROWN.

TREATMENT.

THE USE OF SILK LIGAMENTS IN THE TREATMENT OF (452) INFANTILE PARALYSIS. ROBERT SOUTTER, *Boston Med. and Surg. Journ.*, June 4, 1908.

DR SOUTTER here contributes an interesting article on the above method, first suggested by Lange of Munich. Short notes of a number of cases successfully operated on by the author are also given. As recommended by Robert Jones, Soutter strongly insists on no operation being undertaken until massage, electricity, and careful muscle training have been carried out for several years, in order to give the apparently paralysed muscles a chance of recovering their function. The use of silk ligaments is indicated in a number of different cases. Thus it may be done (1) in addition to transplantation of non-paralysed tendons when the deformity is great or where the transplanted muscle cannot reasonably be expected to correct the deformity, although it may take up the new motion; (2) in cases where transplantation cannot be done owing to the distribution of the paralysis. Here the unparalysed muscles are thrown out of commission; by the use of silk ligaments the distortion is corrected, and in this way from half to all of the function of the unparalysed muscles may be utilised; (3) in a few cases of total paralysis; most of such cases, however, are best treated by arthrodesis; (4) the method may also be tried before proceeding to tendon transplantation in order to give the apparently paralysed muscles a further chance of recovery.

The silk causes the formation of tough fibrous tissue around it which not only reinforces the silk, but is eventually strong enough to take its place. Such ligaments stiffen the joint (ankle) sufficiently to prevent lateral motion, but allow dorsal flexion, which

is so necessary in walking. Soutter uses No. 14, 16, or 20 silk for the ankle. Above, the silk ligament is attached to the periosteum of the tibia, below to the particular tarsal bone indicated—cuboid, scaphoid, etc. To do this, the periosteum of the tibia is split vertically, the edges everted, and the silk introduced by a needle, in and out, on either side. In this way a firm hold above is obtained by the loop of the strand of silk. Below a hole is drilled in the tarsal bone, one of the free ends of the loop passed through it, and then knitted with the other free end. The superficial wound having been closed, the foot is put up in plaster for eight or ten weeks. After this a brace is worn during the day for four months to limit extremes of motion. This is succeeded by a light brace inside the shoe (for long walks only).

A. A. SCOT-SKIRVING.

Reviews

**VORLESUNGEN UEBER DEN BAU DER NERVÖSEN ZENTRAL
ORGANE DES MENSCHEN UND DER TIERE.** Professor
L. EDINGER. Zweiter Band, Siebente Auflage, 1908, S. 334, mit
283 Abbildungen. Price M. 15.

PROFESSOR EDINGER'S well-known text-book on the anatomy of the central nervous system ran through six editions in its original one-volume form, but during the first nineteen years of its existence the subject to which it is devoted had grown so much that when the time came to prepare the seventh edition the author was forced to extend his text to so many pages that two volumes became necessary. The first of these, which appeared four years ago, dealt only with the anatomy of the nervous system of the mammalia; the second volume, which is at present under review, is devoted to the comparative anatomy of the nervous system of the lower vertebrates.

Much that is contained in this volume is entirely new, but knowledge from all available sources has been carefully sorted and incorporated. It is scarcely possible to praise this volume sufficiently or to over-estimate its value, as in it we now possess for the first time a complete account of structure and phylogenesis of the vertebrate nervous system.

The subject is so immense that the author, despite his exceptional experience, found it advisable to avail himself of the aid of authorities on special subjects. Thus the first three chapters, which are devoted to the structure, development, and functions of the peripheral nerves, were entrusted to Professor Froriep; and

Wallenberg is largely responsible for those on the spinal cord and medulla oblongata. Nothing could be more admirable than the manner in which Edinger has fused these pages into his own so as to present to his readers a harmonious whole.

The book is written in the form of lectures; this form allows the author more freedom in expression and description. Its first three chapters deal with the development, classification and connections of the spinal and cranial nerves, and the following two with the spinal cord; the next five are devoted to the anatomy of the medulla oblongata in the aquatic and air-breathing vertebrates; the description of the cerebellum and mid-brain occupies three lectures, the thalamencephalon two, and the last three chapters are devoted entirely to the forebrain.

It is scarcely possible to single out any of the matter of the volume for detailed reference in a short review, but the importance of comparative anatomy in indicating or at least suggesting the nature or function of special parts is well represented by the manner in which the nerves of taste are dealt with. Taste-buds, which in air-breathing vertebrates exist only in the mucous membrane of the mouth and pharynx, extend to the surface of the head and even to the trunk in fishes. In this class these organs, or at least structures with a chemo-receptive function, are supplied by a special group of viscerosensory fibres which owing to their relatively large bulk can be easily traced; they run in the sensory facial and in the glossopharyngeal and vagus nerves, and are connected with the Kiemenspaltenorgane (Froriep), and terminate in a specialised centre of the medulla, the lobus visceralis. In the amniota the ganglion geniculatum, the ganglion petrosum, and ganglion nodosum are the only remnants of the Kiemenspaltenorgane, and it is consequently in the nerves connected with them that the path of taste should be sought. The primary taste centre, on the same line of argument, probably lies in or near the fasciculus solitarius, which is the homologue of the lobus visceralis.

The eighty pages devoted to the structure and evolution of the forebrain will be of general interest; the lucid description and the numerous illustrations, many of them diagrammatic, combined with the author's unique authority, should make this section extremely welcome to all who are interested in the origin and the mode of evolution of "the organ of intellectual life."

The majority of the illustrations are new and original; the greater number of them are reproduced from drawings or reconstructed models, but there are many diagrams.

The volume concludes with a bibliography which includes almost all the important contributions to the development and the comparative anatomy of the nervous system.

GORDON HOLMES.

**THE ANATOMY OF THE BRAIN AND SPINAL CORD, WITH
SPECIAL REFERENCE TO MECHANISM AND FUNCTION.**

HARRIS E. SANTEE. Fourth edition, revised and enlarged.
Pp. 453, with 128 illustrations. London: Appleton, 1908.

THE aims of this volume may be expressed in a paragraph from the author's preface: "The special objects held in view throughout the book are the location of functional centres and the tracing of their afferent, associative, and efferent connections. Particular emphasis is laid upon the origin, course, termination, and function of conduction paths. . . . Function is everywhere correlated with structure; and so far as present knowledge permits, the function of each group of neurones is given in connection with its anatomical description." The author adheres to this programme throughout the greater part of his volume.

Nothing would be more welcome than an anatomical text-book constructed on these lines, in which a sufficiently full and accurate account of the structure of the nervous system would be combined with an intelligent appreciation of the functions of the different parts described; such a book would be an invaluable basis for clinical study.

But anatomy as a science must deal with concrete facts alone, and when such an attempt is made special care must be taken to avoid the danger of constructing anatomical diagrams to explain the mechanism of functions. Into this error, it appears to us, the author has here and there fallen. It is, for instance, repeatedly stated that the trunks of the facial nerves receive fibres from the oculomotor nuclei for the supply of the upper facial muscles, and others from the hypoglossal nuclei for the orbicularis oris. This hypothesis, it is true, was at one time put forward to explain certain clinical observations; but what anatomical evidence has ever been produced of such connections? And what evidence, either clinical or experimental, is there that "a destructive lesion in the nucleus of the seventh nerve causes inferior paralysis of the face, the frontalis, corrugator, orbicularis oculi, and orbicularis oris not being affected"?

The attempt to correlate structure with function has not produced a happy result in the sections that deal with the "sensory or afferent paths." In addition to a direct route—the median fillet and the spino-thalamic tracts—there is also, according to Santee, an indirect route for sensation, formed by the spino-cerebellar tracts and the external arcuate fibres from the dorsal column nuclei to the corpora restiformia, through which sensory impulses run to the cerebellar cortex; thence they are transmitted through the superior cerebellar peduncle to the red nuclei and optic thalami, from which they are conducted by the cortical fillet to the cortex. This view is put forward regardless of the fact that all recent experimental and clinical observations on the functions

of the cerebellum emphatically negative the theory that this organ is in any way concerned with conscious sensation. The author does not adduce any evidence that the spino-cerebellar tracts convey impulses of any form of sensation, though he attributes to the dorsal the conduction of the "muscular and tactile senses, chiefly from the viscera," and regards the ventral as a "path conveying pain, temperature, and tactile impressions." It is regrettable that there is no discussion on such controversial points, and that the facts or the authority on which such important statements and conclusions are based are not quoted.

Despite these and other points against which adverse criticism might be directed, the author may be congratulated on the volume he has produced; it is eminently practical, and should prove of considerable service to the student of clinical neurology. It contains 128 illustrations, many of which are printed in colours; the majority have been judiciously selected from other text-books; but unhappily the author cannot be congratulated on the success of his artist in the execution of many of the original figures from microscopical sections.

GORDON HOLMES.

MODERN CLINICAL MEDICINE—DISEASES OF THE NERVOUS SYSTEM. Sidney Appleton, London, 1908. Price 28s.

THIS work is an authorised translation of the articles on nervous diseases which have appeared at various times since the opening of the century in *Die Deutsche Klinik*. The names of the original contributors are sufficient guarantee of the quality of the work, although in a few instances some of the very latest views may not be referred to. The actual translator's names are not given; we need only say that the translation appears to be well done. One word of warning, however, must be given to those who might seek this volume as a reference text-book for nervous diseases. It can scarcely claim to be a complete text-book of nervous diseases, for, to mention only some of the more outstanding, we find no special consideration of such conditions as meningitis, poliomyelitis, brain tumours, chorea, paralysis of III., IV., and VI. nerves. They may be referred to, incidentally, it is true, under such headings as pathological histology and general diagnosis, but in a general text-book more than that is expected.

J. H. HARVEY PIRIE.

VEREINIGUNG FÜR GERICHTLICHE PSYCHOLOGIE UND PSYCHIATRIE IN GROSSHERZOGTUM HESSEN.

Viertes Heft. (Der Alkoholismus. Seine strafrechtlichen und sozialen Beziehungen. Seine Bekämpfung.) BALSER, AULL, and WALDSCHMIDT. Halle: Marhold, 1908. M. 2.

THIS work consists of three parts. The first, by Medizinalrat Kreisarzt Dr Balser of Mainz, is entitled *Zur forensischen Bedeut-*

ung des Alkoholismus, and deals with those forms of alcoholic poisoning most frequently met with in medical jurisprudence. Among the subjects treated (with illustrative examples) are atypical forms of alcoholism, dipsomania, epileptic conditions and their relation to alcoholism, delirium tremens, acute alcoholic hallucinatory insanity, the bearing of alcoholism on divorce, and certain statutes in the law of Germany.

The second article, entitled *Alcohol und Verbrechen*, by Assessor Aull, Offenbach a Main, discusses the direct and indirect bearings of alcoholism on crime. Numerous interesting tables are given, showing comparative statistics of the consumption of alcohol in Germany for different years, with the number of criminal convictions for corresponding periods. Days of the week, months of the year, and various districts in the German Empire are treated in the same manner, and a striking correspondence is brought out between increase in drinking habits, special facilities of drinking, etc., and the amount of crime. The effects of alcoholism on heredity and family life are emphasised.

Die Behandlung der Alkoholisten, by Dr med. Waldschmidt, Charlottenburg, forms the third and last paper. Many aspects of the question of treatment are discussed. The complete withdrawal of alcohol—while it is a *sine qua non* of treatment—is not alone sufficient. There is a deceptive stage of apparent recovery, and the patient must be carried beyond this before he can be considered cured. The risk is that the patient leaves the institution where he is being treated too soon. A long period of treatment is essential. Voluntary entrance into a Home should be encouraged.

A. HILL BUCHAN.

BOOKS AND PAMPHLETS RECEIVED.

Spitzka. "A Study of the Brains of Six Eminent Scientists and Scholars belonging to the American Anthropometric Society, together with a Description of the Skull of Professor E. D. Cope." Amer. Philos. Soc., Philadelphia, 1907.

Bresler. "Die Willensfreiheit in moderner theologischer, psychiatrischer, und juristischer Beleuchtung." Marhold, Halle, 1908, M. —80.

Liepmann. "Drei Aufsätze aus dem Apraxiegebiet." Karger, Berlin, 1908, M. 1.50.

Kurt Mendel. "Der Unfall in der Ätiologie der Nervenkrankheiten." Karger, Berlin, 1908, M. 5.

Anton. "Vier Vorträge über Entwicklungsstörungen beim Kinde." Karger, Berlin, 1908, M. 1.80.

Ludwig Bach. "Pupillenlehre. Anatomie, Physiologie, und Pathologie, Methodik der Untersuchung." Karger, Berlin, 1908, M. 12.

Review of Neurology and Psychiatry

Original Articles

CASES ILLUSTRATING THE COURSE AND PROGRESS IN DISSEMINATE SCLEROSIS.

By W. B. WARRINGTON, M.D., F.R.C.P. Lond.,

Physician to the Northern Hospital and Eye and Ear Infirmary, Liverpool ;
Lecturer in Clinical Medicine and Neuropathology in the University.

OF the four cases I here record, the first three will, I think, be admitted to be ordinary disseminate sclerosis. May W., as far as could be ascertained, had previously shown no signs of the disease until the rapid development of extensive palsy occurred. The presence of old optic atrophy in Mrs X. brings her case into line with those recently described by Dr Williamson (1). Her illness commenced with a very rapid onset of palsy of considerable extent, followed by an apparent complete recovery.

Such cases are no doubt not very common, but are well recognised, though hardly perhaps yet sufficiently appreciated in general by the profession.

The history of the third patient, D. R., is instructive as showing at how late a period of the disease marked though temporary recovery may yet occur.

They certainly indicate how cautious one should be in giving too unfavourable a prognosis when the diagnosis has been made. Some time ago I saw, with Dr M'Cann of this city, a lady aged about 45, who gave a history of recurrent attacks of paralysis since she was 24 *years of age* ; they had all been followed by a

practically complete recovery, and had been considered to be functional in nature. Such an extended duration afforded strong presumptive evidence against there being any gross organic disease, yet on each of the several occasions of my visits there was a distinct bilateral extensor toe response with absence of the abdominal and epigastric reflexes, and, moreover, this attack evidently was of greater severity than its predecessors, and many months elapsed before the lady was able to walk without some support.

The last case, that of Mr Z., raises the question of diagnosis. The course of the disease, the paralysis of the thoracic muscles, the wasting of the muscles of one arm, and the disorder of stereognostic sense are not features ordinarily seen in true disseminate sclerosis, yet in the interesting case recently recorded by Karl Wegelin (2), where the profound disturbance of sensation and atrophy of muscles might have led to the diagnosis of a "myelitis," the post-mortem disclosed what Wegelin considers to be patches of sclerosis which differed in no single respect from the classical description of the patches in disseminate sclerosis.

Professor J. Hoffmann (3), in an address delivered in 1901, gives a formidable list of twenty-four morbid conditions from which a differential diagnosis is to be made, and amongst these he places multiple myeloencephalitis acuta et subacuta, between which and disseminate, he says, "sind die Grenzen flüssig."

Oppenheim (4), in his valuable article on "Encephalitis" in Nothnagel's System, mentions disseminate sclerosis as one of the events in which myeloencephalitis may terminate, and Edward Müller considers that the anatomical feature of disseminate sclerosis may be the final stage of quite different processes.

Pathological studies can alone decide these questions. Clinically, however, cases resembling that of Mr Z. differ entirely from acute hæmorrhagic disseminate myelitis, nor do they much resemble those illnesses which follow fairly definitely the infective diseases, and which were the subject of Sir Thomas Barlow's (5) Presidential Address to the Neurological Society.

The essential clinical point of distinction seems to be the variability of the symptoms. The disease may begin in an acute manner and its course be characterised by acute exacerbations. In the myelitic and encephalitic processes the history rather is

that of a steady process either to recovery or death, or to a permanent and fixed residual defect.

SHORT SUMMARY OF CASES.

1. May W., æt. 18.—Rapid development of paralysis within three weeks, with nystagmus and diplopia and great mental disturbance. Gradual recovery in six weeks. Now can walk a fair distance.

2. Mrs X., æt. 40.—Optic atrophy known to exist for two years without other symptoms. Attack of sudden giddiness followed by marked paralysis, obtaining a maximum in about fourteen days. Apparent complete recovery. Can walk four miles.

3. D. R., girl æt. 22.—Total duration of illness known to be about eight years, culminating towards the end of the seventh year in spastic ataxic paralysis, with inability to stand. Marked recovery for nearly a year, then rapid onset of bulbar symptoms, ending in death within a week from their onset.

4. Mr Z., æt. 25.—Appears to have had deficient vision and attacks of giddiness two years before onset of present illness, twelve months ago, and three months after an acute illness loss of power rapidly developed in left-sided limbs. Nystagmus, pallor of right disc. Intention tremor right arm; increase in the palsy of all the limbs, paralysis of intercostal and abdominal muscles, and loss of the knee jerks. Some disorder of sensation later, improvement in the upper limbs, marked spastic paralysis of lower limbs remains, yet able to stand. Duration of acute symptoms threatening life about three weeks.

May W., æt. 18, under the care of Dr Sclater of Liscard, was first seen by me on August 30, 1907, a young lady who until her present illness had been a thoroughly healthy girl.

About three weeks previously she had noticed a numb feeling in both hands, and occasionally her grasp became weak, so that she dropped objects. The menses were due about this time, and with their occurrence the patient felt unusually ill, and in a few days had to go to bed feeling "feverish" and out of sorts. She remained in bed a few days, and on getting up felt weak in her lower limbs.

Ten days before my visit the legs suddenly "gave way," and

she had to be carried to bed. She then complained of double vision, the memory became very impaired, so that she could not recall events which had happened a few hours before.

Loss of power in the right upper limb now set in.

When seen by me, the general nutrition and appearance was that of a healthy young woman, but the childish, excitable manner of speaking, and the marked deficiency in memory arrested attention. The right upper limb was markedly paralysed, lying flaccidly by the side, slight flexion of the fingers and some little movements of the shoulder girdle alone being possible.

There was also slight deficiency to light touch over most of the limb.

The left upper limb was also weak, and though it could be moved, the patient was unable to feed herself, and distinct tremor was present in attempting to touch any object. The triceps, radial and ulnar jerks were equal and brisk.

Both lower limbs were markedly paretic; the heels could just be raised off the bed; no loss of sensation was discernible. Both knee jerks were brisk, the $R > L$. The ankle jerks present, but no clonus. The right plantar reflex was distinctly of the extensor type; the left sometimes flexor, sometimes extensor.

The abdominal and epigastric reflexes were absent.

There was no affection of bladder or rectum.

Distinct lateral nystagmus was present, but though the patient complained of double vision there was no obvious impairment of movement of the eyeballs. The optic discs were rather diffused; vision seemed good, and equal in both eyes.

I then lost sight of the patient, but am told that the mental symptoms increased, she could not remember the doctor's visit for more than a few minutes, often there was marked depression and emotional disturbance. She spoke of throwing herself downstairs, or of killing herself with a knife, etc.

These marked symptoms and the paresis gradually cleared away, so that in six weeks' time she was able to get out of bed and move about with help.

Diplopia, choking sensations, and apparent difficulty in swallowing were troublesome symptoms.

The lady was last seen by me on July 22nd of this year. Judging from the private account of her previous state, I judged

that a marked alteration had taken place in her disposition and mental capacity. She behaved in a childish, frivolous manner, making silly jokes, and had largely lost power of attention and concentration. The depression had, however, vanished, and the memory was considerably better.

She was able to walk about without help, though in a jerky manner, advancing the legs in an abrupt ataxic manner. The power was fairly good, and her parents told me she could walk a mile or more without a rest. The upper limbs were fairly strong, though slight "intention tremor" was present in both.

There was no loss of sensation. Ankle clonus and bilateral extensor toe reflex were present. The abdominal and epigastric reflexes remained absent.

Nystagmus was marked. The optic discs were normal, and vision = $\frac{6}{6}$ with both eyes. A hesitancy and difficulty in passing water was not infrequent, once there had been retention for thirty-six hours. Occasionally, when in bed, the urine had been voided involuntarily.

Mrs X., aged 40, was first seen by me on June 1st, 1907. The lady was the mother of several healthy children and had enjoyed excellent general health. Two years before she had suddenly lost sight in one eye, and had consulted an eminent ophthalmic surgeon, who stated there was optic atrophy. Nothing else was noticed, however; the vision was said to have improved, and all went well until three weeks before I saw her, when on getting out of a tramcar she experienced an acute giddiness and staggering; she went home at once and these symptoms passed away. About a fortnight later, difficulty in standing or walking became apparent, and a numbness over both left limbs was complained of.

On examination I found the patient compelled to lie in bed on account of weakness in the limbs. There was distinct flaccid paresis of both lower limbs, more so in the left than the right, and also of the left arm, and on movement slight but distinct intention tremor. The plantar reflex was of the extensor type on both sides. The eyes showed distinct though slight lateral and vertical nystagmus. The left disc was markedly atrophied.

On June 14 the tremor had nearly disappeared, and power had returned to a considerable extent in both left-sided limbs, but the paresis of the right lower limb had increased and pain

about it had become a somewhat obtrusive fact, but these symptoms gradually disappeared, and a month later she could walk about with support, though diplopia was present. I did not see my patient again till January of this year, when I accidentally met her in a public reception of medical men; she appeared quite well, and I venture to think that had I told many of my professional colleagues that the lady suffered from disseminate sclerosis, my diagnosis would have been received with some incredulity. She told me she could walk four miles and over without trouble, and I am glad to learn that this state of affairs still continues.

Miss D. R., æt. 22, under the care of Dr Medwyn Hughes of Ruthin, was first seen by me on 9th February 1904 on account of difficulty in walking of five years' duration. The gait was peculiar, the limbs being advanced in an abrupt, uncertain and somewhat ataxic manner. The patient was able to walk a fair distance, and the power of the various groups of muscles was good. Both knee jerks were brisk, ankle clonus on the right side, not on the left. There was slight impairment of sensation in the lower limbs extending upwards as far as the lowest dorsal zone.

She also complained of a numbness in the hands, and mentioned that sometimes during the last four years there had been a hesitancy and difficulty in passing urine.

The optic discs were normal, and vision good, there was no nystagmus or diplopia and no tremors were present. The general nutrition was good, but a distinctly enlarged thyroid gland was found and there was some exophthalmos, but no other eye signs of Graves' disease. The pulse, however, was 100, and Dr Hughes told me it was usually too fast.

Miss R. was next seen a year later; the difficulty in walking had increased, so that support was required, the extensors of the feet being particularly paralysed. A typical extensor toe reflex present on both sides. Lateral nystagmus had appeared, and the temporal half of the right optic disc was pale. There was a distinct "intention tremor" in both upper limbs.

Enlargement of the thyroid, prominence of the eyes, and a pulse rate of 120 were still obvious features.

The patient was not seen again until February 1907. The spasticity and loss of power in the lower limbs had markedly increased, and she could only stand with support. Pallor of the right disc, with $V = \frac{6}{12}$. Nystagmus, slight inco-ordinate

intention tremor, absence of abdominal reflexes, and an occasionally staccato speech, were obvious symptoms of disseminate sclerosis.

The prominence of the eyeballs and enlargement of the thyroid were still as before, but the pulse rate had dropped and was rarely over 90. In addition to the inco-ordination of gross movement in the upper limbs, there was also a fine, delicate tremor noticeable in the hands, like that of Graves' disease.

The patient was admitted into hospital, and remained there until November 23, 1907. No improvement occurred, and the patient was discharged barely able to move about, even when helped by the nurses.

I have recently learned the history of the termination of her illness. Some time after leaving the hospital she began to improve, and gradually became so strong that she was able to walk about without support, and could go upstairs alone, and do "fancy" work. The exophthalmos and tremor disappeared. The end came suddenly; in the beginning of April of this year, pain in the back and inability to walk or stand rapidly set in. The patient had to go to bed. She soon became delirious in a quiet, rambling way, then there was absolute blindness for six hours, followed by some return of vision. Two days before death, difficulty in swallowing, paralysis of the tongue, and complete paralysis of all four limbs were present. The duration of these terminal events was about one week.

Mr K., æt. 25, was brought to me by his brother, a medical man, on September 9, 1907. A year ago, when in Canada, he had a severe attack of "gastro-enteritis," accompanied with sharp fever; he was in bed ten days, but appears to have quickly convalesced, and returned home to England apparently quite recovered. Last Christmas—*i.e.* about three months after the acute illness—loss of power was observed in the left leg, so that in April he was no longer able to work. After a time the weakness disappeared to such an extent that he was able to walk, and what caused him to seek advice was a rather sudden weakness in the left arm, accompanied by tingling sensations in both upper limbs and in the left leg.

He then told me that he had observed deficiency in vision in the right eye for two years, and had been subject to attacks of giddiness.

On examination he presented the symptoms of profound disturbance of the nervous system. He was unable to stand without support, the left lower limb was almost completely paralysed, extension and flexion of the foot being just possible. The power in the right leg was fairly good. Both knee jerks were exaggerated, ankle clonus on the left side, and a typical extensor toe on both. The abdominal and epigastric reflexes were absent.

The left upper limb was also weak, with some wasting, especially in the small muscles of the hand.

Slight "intention-tremor" could be elicited with the right arm. There was marked lateral nystagmus; the right pupil was $>$ left, right vision = $\frac{6}{18}$, left $\frac{6}{8}$, and there was distinct pallor of the right disc on its temporal side. No diplopia or paresis of external eye muscles.

The speech was perhaps a little slurring in character.

There had been no bladder trouble.

I am indebted to Dr K. for the remainder of the clinical history:—

The patient was put to bed, and in two days the weakness of the upper limbs had increased to such an extent that the patient had to be fed by the nurse, and the abdominal muscles appeared palsied. On September 27th palsy of right leg was noted, paralysis of the intercostals, flaccidity of the abdominal muscles, and loss of the knee jerks. Two days later there was alarming difficulty in breathing, with complete paralysis of thoracic movements. On October 4th he was described as lying in bed unable to change his position, the left arm atrophied, and scarcely able to be voluntarily moved. The right arm had some power, but little co-ordination in its movements. There was also a distinct affection of sensation, so that he could not distinguish between the blankets and sheets of his bed-clothes by touch. Words were spoken in a slurring, indistinct fashion. Owing to the lack of respiratory force he could only speak a few words, not more than five, without a pause.

From time to time there was a paresis of the eye-muscles.

The abdominal reflexes and knee-jerks were absent.

On the 30th November, Dr K. informs me, the patient had steadily improved. He could draw up and extend both legs. The knee jerks had returned. The hand and shoulder could be

moved with fair power. The inco-ordination had also disappeared, but there was still some astereognosis; for example, the patient could not recognise a sixpence, or distinguish it from a collar-stud or thimble. On several occasions a transient right-sided facial palsy had been noticed. The thoracic and abdominal muscles were still very weak. The general health and mental power remained unimpaired.

On May 11th he had so far improved as to be able to sit up in a chair. Every now and then marked inco-ordination in the upper limbs was present, but this was not constant. Sometimes the head and neck were seized with nodding, shaking movements. The knee jerks just obtainable, double ankle clonus, and extensor toe reflexes present. The speech was clear, and nystagmus less marked.

The unfortunate gentleman about this time had a second attack of appendicitis, and his medical attendant thought he was going to die; however, he pulled through well enough, and now can use his arms and hand with strength and efficiency, shooting with a toy pistol straight enough to get "bull's eyes," and can work a typewriter, but cannot write, for a violent jerk carries his pen off before three letters have been formed. The muscles of the left shoulder and arm are somewhat wasted. Sensation and stereognostic sense now normal. There is still the curious tremor, nodding and jerking of the head and neck. The thoracic expansion remains feeble, and seems to explain the slowness of speech, which, however, has no scanning or staccato features.

There is a distinct pallor of the right disc, and less so of the left, and some failure of vision. Nystagmus has disappeared. Knee jerks just obtained; abdominal reflexes absent. Typical double ankle clonus and extensor toe reflex.

He can now stand, but spasm of the muscles of the feet tends to upset him. Individual movements of muscle groups can be fairly well performed.

The action of both bowels and bladder are at times precipitate.

Mr Z.'s appetite, general health, and mental power are in no way impaired.

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THREE CASES OF HEMIPLEGIA FOLLOWING SCARLET FEVER.

By J. D. ROLLESTON, M.A., M.D., Oxon.,
Assistant Medical Officer, Grove Fever Hospital, London.

THE rarity of hemiplegia in scarlet fever is illustrated by the fact that the present three cases are the only ones that have occurred among 10,781 consecutive cases of scarlet fever admitted to the Grove Hospital between August 1899 and December 31, 1907. A prolonged search through literature has enabled me to add 63 more, thus making a total of 66 cases. It is well known that hemiplegia may follow any infectious disease. Forty-six cases have recently¹ been collected by Smithies in typhoid fever, and 65 cases in diphtheria by myself. When one considers the greater prevalence of scarlet fever compared with diphtheria or typhoid fever, it is obvious that hemiplegia is much less common in scarlet fever than in the other two diseases.

It is well to insist on the rarity of hemiplegia in scarlet fever, as the statements of some neurologists are somewhat misleading. More confidence should be given to pædiatrists, who see much of scarlet fever, than to those who are likely to be consulted only for its nervous sequelæ. It is instructive to compare the words of Sir W. Gowers: "Sudden cerebral hemiplegia (in scarlet fever) is not infrequent," with the following quotations: "Nervous complications and sequelæ are seen less frequently after scarlet fever than with most of the infectious diseases of such severity" (Holt). "Les paralysies dans le cours de la scarlatine sont excessivement rares" (Moizard). "Eine Betheiligung des Gehirns am Scharlachprocesse kam mir nur selten vor" (Hench). Similar testimony is given by Landouzy in his well-known monograph, "Des paralysies dans les maladies aiguës," in opposition to Gubler, who regarded paralysis in scarlet fever as fairly frequent. In this connection it may be said that some authorities have a mistaken tendency to ascribe too large a number of cases of cerebral palsy to infectious disease. Sachs and Spiller have rightly protested against this view. In striking

¹ Two additional cases have since been recorded by Barié and Lian, and Laignel-Lavastine (v. abstract in *Review of Neurology and Psychiatry*, 1908, p. 37).

contrast to Marie and Jendrassik, who were of opinion that infectious disease was the only cause of infantile hemiplegia, Sachs states that the appearance of this palsy after infectious disease could be affirmed with certainty in only 20 per cent. of his 225 cases.

In discussing the ætiology of hemiplegia in scarlet fever, it is interesting to note that the occurrence of this paralysis is by no means confined to young children. This is clearly shown in the following table:—

TABLE 1.—Ages and sexes of scarlet fever patients at the onset of hemiplegia.

Ages.		Male.		Female.
0—1	...	0	...	1
1—2	...	2	...	1
2—3	...	3	...	4
3—4	...	3	...	2
4—5	...	3	...	1
5—6	...	4	...	3
6—7	...	1	...	7
7—8	...	4	...	5
8—9	...	1	...	2
9—10	...	0	...	1
11—12	...	1	...	0
12—13	...	1	...	0
13—14	...	0	...	1
14—15	...	1	...	0
15—16	...	0	...	2
16—17	...	0	...	1
20—21	...	1	...	0
24—25	...	0	...	1
		—		—
		25		32

In 9 the ages and sexes were not recorded. Thus 20 cases occurred in the first quinquennium, 28 in the second, 4 in the third, and the remainder in older patients. Right hemiplegia occurred in 43, left in 15. In Muls's case there was crossed hemiplegia (left facial palsy, with paralysis of the right upper and lower limbs). In 7 no details were given. Hemiplegia took place at the following dates:—In the first week of scarlet fever

5 cases, in the second 7, in the third 5, and in the fourth to sixth week 12. In cases where no exact date is stated it is said to have occurred in convalescence in 18 cases. In 19 no date whatever was stated. Recovery took place in 49, but in only 17 cases was it complete. In the majority contractures and atrophy supervened. Henoch's case is remarkable in that the contractures disappeared at the end of a year. Among other sequelæ athetosis is mentioned in the cases of Barlow, Dejerine, Fisher, Fürbringer, Montgomery, and Pastore; chorea in Fürbringer's, Kennedy's, and one of Osler's cases; recurrent convulsions in those of Barlow, Bernhardt, Dejerine, Heubner, Lewis Smith, Montgomery, and Wallenberg; and failure of the memory and intellect in the cases of Bernhardt, Bohn, Condie, Hughlings Jackson, Osler, and Wallenberg.

Death occurred in 11, in 8 of which there was an autopsy. In 3 of the latter, however, death took place many years after the onset of hemiplegia, being due in one case to pneumonia at fifty-four (Dejerine), and in others to phthisis at nineteen and twenty-seven (Bernhardt and Taylor). In 6 the issue of the cases was not recorded.

In 28 of the cases of right hemiplegia the paralysis was associated with aphasia. It is of historical interest that the two phenomena were combined in the earliest case recorded, that of De Haen in 1760.¹ In 3 it is stated that there was no aphasia, but one of these patients (Dr F. Taylor's case) was left-handed. In the great majority of cases in which details are given, *i.e.* in 22 out of 30, the initial scarlatinal attack was severe. In the remainder, wherein may be included the three cases to be described, it was mild. In some of the recorded cases another infectious disease had recently preceded scarlet fever, *e.g.* diphtheria in the cases of Heubner and of Fyshe and Hunter, and whooping-cough in the cases of Walker and one of Osler's patients.

Since diphtheria and whooping-cough may each be followed by cerebral palsy, it is impossible to exclude them from the causation of the hemiplegia in these cases. In 28 cases, including Cases II. and III., hemiplegia was associated with scarlatinal

¹ Puella 16 annorum febre scarlatina correpta mense Septembri anno 1760, dein convulsione 7 Octobri latere dextro paralytica facta est, et perfecte aphonya.—Quoted by Imbert-Goubeyre, *loc. cit.*

nephritis. In the remaining 38 nephritis either did not occur or was not recorded.

As already stated, autopsies were held in 8 cases. Cerebral hæmorrhage was found in 3 (Dejerine, Fürbringer, Southard and Sims), embolism in 1 (Taylor), thrombosis in 1 (Alexeff), sclerosis in 1 (Neurath), and atrophy in 2 (Bernhardt and Taylor). Out of the fatal cases on which no autopsy was held hæmorrhage was diagnosed in 1 (Sufrin), embolism in 1 (Case I.) and uræmia in 1 (Case III.). In the cases which recovered the diagnosis of hæmorrhage was made in 4 (Henoch, Luukonen, Montgomery, and Sufrin), of thrombosis in 5 (Addy, Baginsky, Bazin, and Ferrier), of embolism in 11 (Barlow, Cheripin, Freud, Fyshe and Hunter, Hughlings Jackson, Kennedy, Osler, Rosenberg, and Wallenberg), of acute encephalitis in 3 (Heubner, Muls, and Pastore), and of uræmia in 1 (Case II.). One of Osler's cases was unique in that the hemiplegia was of surgical origin. A cervical abscess following scarlet fever gave rise to ulceration of the right carotid, necessitating ligature in a girl of six. Left hemiplegia followed, and was still present at the age of twenty-four.

Table II. shows that apart from uræmia cerebral embolism has been regarded as the commonest cause of hemiplegia in scarlet fever. However, now that more attention has recently been given to acute non-suppurative encephalitis as a cause of cerebral palsy, more cases may be found due to this cause than has hitherto been supposed.¹ In 18 cases no mention is made of a cardiac lesion, but the occurrence of nephritis alone was noted. With the exception of three cases recorded by Alexeff, Bohn, and Semple respectively, in which complete recovery took place, the paralysis was permanent. It is highly improbable, therefore, that the hemiplegia, except in the three cases mentioned, could have been due to uræmia, since almost invariably uræmic paralysis is of transient duration. It is far more likely that coexistent hæmorrhage or embolism due to an undetected cardiac lesion was the cause of the hemiplegia in the other cases. If this supposition is correct, only three cases of uræmic hemiplegia in scarlet fever have hitherto been recorded, to which number Cases II. and III. may now be added.

¹ Rhein has recently recorded a fatal case of scarlet fever in which acute encephalitis was found at the autopsy. Death had been preceded by convulsions, but no paralysis had occurred.

In Walker's case, in which the hemiplegia occurred on the first day of disease, the paralysis was attributed to the direct action of the scarlatinal virus on the cerebro-spinal system. In Bassette's case the condition was regarded as a neuritis, though the existence of a mitral lesion and the disturbance of speech make cerebral embolism more probable.

TABLE II.—Showing causes of hemiplegia in 66 cases :—

Cerebral embolism . . .	13 cases
„ hæmorrhage . . .	8 „
„ thrombosis . . .	6 „
Encephalitis . . .	3 „
Cerebral atrophy . . .	2 „
„ sclerosis . . .	1 case
Nephritis. Uræmia . . .	17 cases
Neuritis . . .	1 case
Ligature of carotid . . .	1 „
Toxæmia (Walker's case) . . .	1 „

53

No cause was assigned in 13 cases.

CASE I.

A woman, aged 24, a milliner by occupation, was admitted to hospital on July 6th, 1904, on the sixth day of an attack of scarlet fever. For the last few weeks she had been treated for anæmia, but there was no history of heart disease. She stated that she had already had scarlatina in 1888.

On admission there was a fading punctate erythema on the trunk. On the limbs the rash was brighter. Examination of the heart revealed a systolic murmur localised to the apex. By July 8th the rash had faded. Characteristic desquamation followed. The temperature, which was 101.6° on admission, did not sink below 98.8° , but remained partly continuous and partly intermittent during the rest of her stay in hospital. On the 11th she vomited three times, and complained of pain in the region of the spleen. There was some tenderness over that area, but no enlargement of the organ could be detected till the 18th, when its lower border could be felt one finger's breadth

below the costal margin. On the 13th the urine, which had hitherto been normal, showed a cloud of albumin, and on the 18th hæmaturia developed. Blood disappeared from the urine the following day, and subsequently was present only on the 22nd and 23rd of August. Albuminuria, however, persisted.

On July 15th petechiæ appeared on the trunk. On the 22nd and 23rd she complained of pain throughout the left lower limb. On the 24th the pain became localised to the upper part of the calf and then disappeared. The same day the cardiac apex beat was found to be outside the nipple line, the dulness extending $1\frac{1}{2}$ inches beyond it. The systolic murmur was now conducted into the axilla. A few fresh petechiæ were noted on the trunk. On the 27th she woke up suddenly at 1 A.M., muttered something inarticulate, and was unable to move her right arm and leg. Examination in the course of the morning showed complete right hemiplegia. In addition to the motor palsy, anæsthesia was present in the parts affected. The tongue was deviated to the right. Well-marked ankle clonus and Babinski's sign existed in the right foot. There was partial motor aphasia. The cardiac condition was unchanged. On the 28th the aphasia was complete, and the tongue was more deviated to the right. The right lower limb could be moved slightly, movement taking place at the pelvis. Sensation was returning in the right side of the face, right side of the trunk, and right lower limb. There was still anæsthesia but not analgesia of the right arm. On the 30th sensation began to return in the right arm. Ankle clonus was still very readily obtained in the right foot. The aphasia continued as before. On the 31st a few fresh petechiæ were noted on the neck and trunk. She was now able to say a few short words at a time and to read aloud from a book. On August 1st the right arm and leg, which since the ictus had been quite flaccid, were now becoming somewhat rigid. There was still some anæsthesia of the paralysed side. On the 2nd she could speak a little more, but could not distinctly articulate polysyllabic words. Slight impairment of the intellectual faculties was shown (1) by an inability to perform simple sums in mental arithmetic. Though she could multiply 5 by 4, she could not give the product of 12 by 12. (2) By a certain degree of amnesia. She was unable to say more than the first few words

of the Lord's Prayer. (3) By inability to read without soon feeling tired. On the 3rd she was able to write the words "wrote" and "millinery," but when asked to write "haberdashery," "crinoline," or "cashmere," a look of vacuity came over her face, and she was unable to proceed. On the 6th, improvement was shown by her being able to write the words "impossibility" and "haberdashery." She could also recite the whole of the Lord's Prayer with very little prompting, and read to herself longer without being tired.

During the rest of her stay in hospital fresh petechiæ were noted almost daily on the trunk and limbs. The spleen gradually increased in size, and the heart became more dilated. During the last three weeks of her stay she had frequent attacks of severe pain in the abdomen and lower limbs, which yielded only to the injection of morphia. On September 3rd, being free from scarlatinal infection, she was discharged. Her condition was then as follows:—The facial palsy had almost gone. There was still some hesitancy in speech and deviation of tongue to the right. The right arm was quite powerless, and there was some rigidity at the shoulder and elbow. The fingers were kept flexed, but could easily be extended. The right lower limb could be moved fairly freely, but there was some stiffness of the knee. Death, preceded by coma of two days' duration, took place on November 26th.

Though no autopsy was held in this case, the causation of the hemiplegia was obviously cerebral embolism, due to infective endocarditis. Multiple embolism doubtless existed. Not only were the brain, kidneys, and spleen affected, but the attacks of pain in the abdomen and limbs were probably due to the discharge of minute clots from the diseased valves. The only other recorded case of scarlatinal hemiplegia associated with multiple embolism is that of Fyshe and Hunter. Their diagnosis was based on the occurrence of sudden and transient attacks of pain in the abdomen and lower limbs, the sudden onset of Jacksonian epilepsy, transient left hemiplegia, and the presence of blood in the stools. Recovery took place. The history of anæmia in the present case suggests that the heart condition may have been present before the onset of scarlet fever, but in any case the rapid subsequent progress of the disease showed that the acute exanthem considerably aggravated, if it did not actually originate,

the cardiac lesion. Of special interest is the age of the patient. Although, as already stated, scarlatinal hemiplegia is not confined to young children, the only other cases in adults are two reported by Ferrier in soldiers whose ages are not given, and one by Achard in a man of twenty.

CASE II.

A girl, aged 13 years, was admitted to hospital on March 28th, 1905, on the second day of a typical attack of scarlet fever. Characteristic desquamation followed. On the 31st she had some rheumatism in the hands, and from April 4th to April 7th there was slight albuminuria. The heart was not affected. The urine subsequently remained clear till the 18th, when it showed a cloud of albumin, and the patient complained of headache. There was frequent but slight epistaxis. On the 19th the urine contained blood and was scanty (8 oz. in twenty-four hours). The face was puffy, especially round the eyes. The temperature, which had been normal since the 6th, rose to 99° at 8 P.M. A drachm and a half of liquorice powder was given, and the bowels acted freely. On the 20th the headache was less, the face was not so puffy, and the flow of urine had increased to 47 ounces in the twenty-four hours. There was still some epistaxis.

On April 21st, the twenty-sixth day of disease, there was only a faint trace of albumin in the urine, but there was still occasional epistaxis. Nothing unusual was observed till 2.45 P.M. that day, when the nurse first noticed some twitching of the face. When I saw the patient ten minutes later, there was loss of power and sensation in the right arm and right lower limb. Ankle clonus was very readily obtained in the right foot. There was no plantar response. The right conjunctiva was insensitive, the left conjunctival reflex was normal. Though she did not speak, she showed that she was conscious by putting out her tongue and shaking her head when told to do so. At 3 P.M. twitching of the right side of the face began, and spread to the opposite side. Shortly afterwards generalised convulsions supervened with loss of consciousness. Cyanosis of the face was very marked. The heart sounds were rapid and irregular in force and rhythm. About 12 oz. of blood were withdrawn from the right elbow, and inhalations of chloroform were given. A minim of

croton oil in butter was also placed upon the tongue. After the venesection the convulsions ceased, and the cyanosis gradually passed off. By 4.30 P.M. the child was somewhat restless, but was gradually regaining consciousness. The right arm, though not absolutely flaccid, was immobile, while the left could be moved freely. At 4.35 P.M. slight vomiting occurred. The right hallux now gave a decided extensor response. The tongue, on being protruded, was deviated to the right.

At 5.35 P.M. consciousness was regained. The right arm could now be moved freely. She could speak a few words, but not distinctly.

At 6 P.M. Babinski's sign was present in both feet. There was now no ankle clonus. The tongue was still deviated to the right.

At 10 P.M. Babinski's sign was replaced by a flexor response.

The temperature was 100° at 8 P.M., but fell to normal at midnight, and subsequently remained subnormal. The urine record for the 22nd and 23rd was 16 and 15 ounces respectively, but on the following days the output considerably increased, the average amount passed during the twenty-four hours being 40 ounces.

On the 23rd the patient seemed quite herself again. The tongue was no longer deviated to the right, but was still sore where it had been bitten during the convulsions.

The urine became free of albumin on the 28th. Beyond the occurrence of pigmented striæ atrophicæ on the breasts, which were first observed on the 28th, nothing further of note occurred, and the patient was discharged on May 24th, 1905. At the time of writing (December 1907), her father informs me that she has enjoyed the best of health since leaving the hospital.

The prodromal headache and epistaxis, the diminution of the urinary secretion, the presence of blood and albumin in the urine, the transient character of the palsy, and the rapid and complete recovery, justify the diagnosis of uræmic paralysis in this case. In the absence of endocarditis, which could have given rise to cerebral embolism, the possibility of cerebral hæmorrhage was at first suggested by the sudden character of the onset and the condition of the reflexes, but this hypothesis was soon set aside by the rapid disappearance of the paresis, and by the restoration of the reflexes to their normal condition. The

difficulty, and even impossibility, of diagnosing uræmic paralysis from paralysis due to cerebral hæmorrhage, embolism, or thrombosis have been dwelt upon by many writers, who point out that there is nothing characteristic in the onset of uræmic hemiplegia to distinguish it from hemiplegia due to other causes. As was illustrated by the autopsy in Alexeff's case, the mere presence of nephritis does not justify the diagnosis of uræmic hemiplegia. In that case, in addition to nephritis, endocarditis and embolism of the middle cerebral artery were found. Further, it is well known that cerebral hæmorrhage is liable to occur in Bright's disease, and it was to this cause alone that any paralysis was attributed by Lasèque, who, like Addison, denied the existence of uræmic paralysis. The imitative character of uræmia has been happily expressed by Chauffard in the following words:—"En matière de pathologie cérébrale, l'urémie est comme la syphilis et l'hystérie: elle peut tout réaliser ou, du moins, tout simuler."

It is noteworthy that, though there was some diminution in the urinary secretion, there was by no means anuria. As has been pointed out by Dr Rose Bradford in his Goulstonian lectures, and as is exemplified in Case III., rapidly fatal uræmia may occur while the patient is passing quite considerable quantities of urine. Leichtenstern has also recorded cases of scarlatinal nephritis, in which oliguria and hæmaturia were replaced by polyuria with rapid disappearance of albumin, and in which, nevertheless, grave uræmic phenomena supervened.

Provided the patient recovers from uræmia, which is always a condition of considerable gravity, the prognosis of uræmic paralysis is good. The paralysis is usually transitory and leaves no sequelæ. Bohn's case is exceptional, in that the child, who before her illness had been highly gifted, became mentally deficient, though she recovered from the hemiplegia and aphasia.

CASE III.

A girl, aged 11 years, was admitted to hospital on October 19th, 1907, on the third day of a mild attack of scarlet fever. On November 6th, the twenty-first day of disease, a trace of albumin appeared in the urine. During the next few days the albumin increased. On the 10th hæmaturia developed, and the temperature, which had been normal since October 9th, rose to

99.6° at midnight. On the 11th she had numerous fits, followed by loss of power in her right arm and leg. Sensation apparently was not affected. Motor aphasia was present, but she appeared to understand what was said to her. Babinski's sign and ankle clonus were present in her right foot, and the right abdominal reflex was absent. The following day the palsy disappeared. In spite of hot packs, croton oil, lumbar puncture, and phlebotomy, the fits continued. Coma supervened, and death occurred on November 14th, the twenty-ninth day of disease. So far from there being anuria, urine was passed freely during her fits. As much as 30 oz. were collected in the last twenty-four hours.

No autopsy was performed.

The occurrence of aphasia in Cases II. and III. deserves special mention. Uræmic aphasia is rare. Like uræmic paralysis, it is usually of short duration. Riesman in 1902 had collected 29 cases, including two of his own, 15 of which were unaccompanied by paralysis, while in the remainder some form of paralysis existed. It is remarkable that Monod, who, faithful to the doctrine of the time, denied the existence of paralysis in uræmia, should have recorded two cases of uræmic aphasia following scarlet fever. In both aphasia was associated with convulsions, as in Cases II. and III., and in both recovery took place.

The occurrence of aphasia was noted in 25 of the 66 cases of scarlatinal hemiplegia which I have collected. In 14 of the 24 cases there was nephritis, but in 4 of these cardiac lesions were also present, and the aphasia was attributed to embolism (Alexeff, Barlow, Cheripin, Rosenberg). It is probable that the same explanation would account for the aphasia in some, if not in most, of the rest.

Dupré, who in 1894 drew special attention to the existence of uræmic aphasia, recorded a case very similar to those under discussion. A boy, aged nine years, while suffering from scarlatinal nephritis, was suddenly seized with complete motor aphasia, agraphia, and paresis of the right upper limb. Within twelve hours speech began to return, and three days later was completely restored. It may be mentioned that aphasia may occur as an isolated symptom in scarlet fever apart from paralysis, just as, but much less frequently, than aphasia does in typhoid fever.

A case of the kind has been recorded by Brasch, in which aphasia developed at the onset of scarlet fever.

The pathogeny of uræmic paralysis is not yet fully elucidated. Though the occurrence of cerebral symptoms in kidney disease was described by Addison in 1839, uræmic paralysis was practically unknown before the appearance of Raymond's monograph in 1885. It is a remarkable fact that the earlier writers, such as Addison and Laségue, laid special stress on the absence of paralysis as characteristic of uræmia. That such excellent clinicians should have denied its existence is sufficient proof that uræmic paralysis is rare. On the strength of some post-mortem findings the paralysis was at first ascribed to cerebral œdema, but this explanation was contested by others on the ground that in many autopsies no trace of œdema could be found, nor was it clear why in cases in which it had been found a diffuse cerebral œdema should produce a unilateral paralysis. The theory of a toxic paralysis *sine materia* was, therefore, substituted, similar to that which may follow such poisons as carbon monoxide, mercury, or lead. More recently Raymond, on the ground of experimental and clinical facts, states that uræmic intoxication very probably produces an acute or subacute encephalitis. In the immense majority of cases the encephalitis clears up, since most of the patients recover from their hemiplegia, but in a few the lesion persists and entails a permanent palsy.

It is surprising that in spite of the frequency of nephritis in scarlet fever uræmic paralysis should be so rare. Such cerebral symptoms as headache, vomiting, drowsiness, and twitching in scarlatinal uræmia are not uncommon. Generalised convulsions are much less frequent, but are less rare than any kind of paralysis. Ferrand in 1893 attempted to explain the rarity of nervous symptoms in uræmia by saying that a specially sensitive nervous system is required for their development, and supporting this hypothesis by the relative frequency of eclampsia in pregnancy. In 1904, in conjunction with Castaigne, he brought forward clinical and experimental evidence to show that uræmic paralysis is due to old lesions of the cerebral motor zones, which for that reason are less resistant to the mechanical and toxic action of cerebral œdema.

This view is of considerable interest, but needs further confirmation. In the absence of any history or evidence of old

cerebral trouble this explanation of uræmic paralysis can hardly be applied to the cases under discussion.

CONCLUSIONS.

1. Hemiplegia in scarlet fever is rare.
2. It is not confined to children.
3. It is usually due to cerebral embolism, but may also follow thrombosis, hæmorrhage, or acute encephalitis. Under this last heading uræmic hemiplegia may probably be placed.
4. The prognosis of hemiplegia in scarlet fever is generally good as regards life, but unfavourable in respect to complete recovery.
5. Uræmic hemiplegia, which probably occurred in only a few of the recorded cases, is exceptional in that it is almost invariably of short duration and leaves no sequelæ.

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Abstracts

ANATOMY.

**NOTE ON THE COURSE OF THE CEREBELLO-OLIVARY
(453) FIBRES, AS DEMONSTRATED IN A CASE OF TUBERCULOSIS OF THE SPINAL CORD AND MEDULLA.**

T. K. MONRO and LEONARD FINDLAY, *Glasgow Med. Journ.*, July 1908.

A MAN, æt. 22 years, was admitted to hospital suffering from vomiting and pains all over body, but especially in the head and neck, of two weeks' duration. Later on he became unconscious, paralysis of the limbs and diaphragm developed, and he died six days after admission.

Post-mortem, there was discovered general tuberculosis, with a more recent basal meningitis. There was in addition an older tubercular spinal meningitis, most extensive in the lower dorsal and lumbar regions. In the fourth lumbar segment there was situated a tubercular nodule, which, along with the spinal meningitis, had produced an extensive ascending degeneration of the posterior columns, cerebellar, and antero-lateral ascending tracts. Situated in the left half of medulla in the region of the olivary nucleus was another tubercular mass, measuring .75 cm. in diameter, and causing in great part the destruction of that nucleus. In the immediate periphery of that nodule there was degeneration of the left pyramidal bundle, left posterior basis bundle and left restiform body, while on the opposite side there was slight degeneration of the fibres in the hilum of the olivary nucleus, but the restiform body and cerebello-olivary fibres seemed almost entirely quite healthy.

From the above facts the authors conclude that the cerebello-olivary fibres are descending in type. **AUTHOR'S ABSTRACT.**

THE STRUCTURE OF THE AUTONOMIC NERVOUS SYSTEM COMPARED WITH ITS FUNCTIONAL ACTIVITY.
(454)

C. V. A. KAPPERS, *Jour. of Physiol.*, June 30, 1908, p. 139.

THE author considers that the axon-reflex (described by Langley) which passes along motor paths only is the prevailing one in the sympathetic system, and has even been the moving agent in the development of its peculiar structure.

From phylogenetic studies of the cranial nerves we know that

the motor cells migrate from their original position in the direction whence the maximum of stimuli reaches them. In the central nerve system the way is shortened by the shortening of the dendrites; the efferent way, the axis cylinder, is elongated in consequence.

Conversely, in the autonomic system the distance between the motor cells and the centres of stimulation (viscera) is shortened by the shortening of the motor axis cylinders themselves. At a distance where in the somatic system there is only one long axis cylinder, there are here more neurons (two, perhaps sometimes three). By analogy, the most frequent stimulation of the motor sympathetic cells has not been from the spinal cord, but along the axis cylinders themselves, *i.e.* an axon-reflex.

The neurobiotactic principle would explain the absence of sympathetic ganglia (except the ciliary) from the cranial nerves of Selachians. The possibility of stimulation of the sensory endings of the cranial visceral nerves is so much greater in them, owing to the endings being spread out over the gills, etc., that the sensory-reflex has so prevailed over the axon-reflex that no sympathetic system has developed. In higher animals these parts are, like the viscera of the trunk, more protected from strong outside sensory stimuli; the axon-reflex therefore prevails, and a sympathetic system develops. The fact that in all animals a ciliary ganglion is present is in perfect harmony with this explanation, as the ocular part of the trigeminus sensibility, as far as concerns the iris, is carefully excluded from strong stimuli.

Another feature might find its explanation in the neurobiotactic influence of the axon stimulation:—the fact that the more distant from the central nervous system, the greater is the number of motor cells; they increase in number the farther they have migrated, because the nearer the cord the greater still is the influence of the common sensory (spinal) reflex arc, and the smaller that of the axon reflex, because the ganglion cells themselves are only slightly, if at all, transgressible for the axon-reflex.

J. H. HARVEY PIRIE.

THE DIFFERENCES BETWEEN CENTRAL AND PERIPHERAL

(455) FACIAL PARALYSIS AND THE ANATOMICAL BASIS OF THE SAME. (Die Unterschiede centraler und peripherer Facialislähmungen und die anatomische Grundlage derselben.)

CARL HUDOVERNIG, *Neurol. Centralbl.*, Juni 16, 1908, p. 577.

THE well-known differences between central and peripheral facial paralysis are described. The author holds that the reason why, in lesions of the facial fibres above the nucleus, the upper part of

the face escapes is to be found in the circumstances that there is a special centre for this region in the cortex, while in the medulla also the fibres which supply the upper face arise from a distinct part of the facial nucleus.

EDWIN BRAMWELL.

PHYSIOLOGY.

VASO-MOTOR INNERVATION OF THE THYROID GLAND.

(456) (*Recherches sur l'innervation vaso-motrice du corps thyroïde.*)

FRANÇOIS-FRANCK et HALLION, *Jour. de Physiol. et Pathol. gen.*, mai 15, 1908, p. 442.

FROM plethysmographic experiments (the technique of which is given) on curarised dogs, the authors arrive at the following conclusions with regard to the vaso-motor supply of the thyroid:—

1. There are vaso-constrictor fibres for the thyroid in the *rami communicantes* as low as the 9th dorsal, but only very few in the lower three of these. They pass in both divisions of the annulus of Vieussens up to the superior cervical sympathetic ganglion, join the external laryngeal nerve a little below where it leaves the superior laryngeal, at least in part, if not entirely, and so reach the thyroid.

2. There are vaso-dilator fibres in the superior laryngeal nerve.

Stimulation of the recurrent laryngeal nerve gave negative or doubtful results so far as vaso-motor fibres for the thyroid were concerned.

3. Although stimulation of the upper end of the cervical sympathetic cord usually caused a vaso-constriction, a reflex vaso-dilatation could also be produced. This seems to be due to the presence of sensory fibres in the cervical sympathetic coming from the endocardium and inner coat of the aorta. Irritation of the aorta and of the endocardium of the left ventricle near the aortic orifice causes a bilateral thyroid vaso-dilatation. After section of the cervical sympathetic this is not produced. These fibres may possibly play a rôle in exophthalmic goitre.

J. H. HARVEY PIRIE.

ON HYPERTROPHY OF THE PITUITARY BODY IN ANIMALS

(457) **AFTER EXCISION OF THE THYROID.** (*Sur l'hypertrophie de l'hypophyse cérébrale chez les animaux thyroïdectomisés.*)

CIMORINI, *Arch. ital. de Biol.*, T. 48, f. 3, 1908, p. 387.

VARIOUS writers have shown that the functions of the thyroid are distinctly separate from those of the parathyroid. Excision of the thyroid is followed by a chronic disease which ends after a time in

cachexia and death ; excision of the parathyroids results in rapid and violent death from tetany.

In order to determine the origin of the changes which take place in the pituitary body after excision of the thyroid and parathyroids, the writer made numerous experiments on the dog and the rabbit, using every possible care in the technique of his operations. He comes to the following conclusions:—

(1) Hypertrophy of the pituitary body after excision of the thyreo-parathyroid apparatus is due to the removal of the thyroid glands, not to loss of the parathyroids.

(2) The data furnished by histological examination of this hypertrophy are of a specific character, owing to the presence of special cells, notable mainly for their large size, and are thus distinguished from the appearances found on histological examination of hypertrophy due to castration.

(3) The formation of these elements should in all probability be attributed to the increase in functional activity in a particular kind of pituitary cells, which cannot be clearly differentiated in normal conditions or in hypertrophy due to castration, but which become evident by their increase in size after removal of the thyroid.

A. N. BRUCE.

THE ACTION OF THE ACTIVE SUPRARENAL PRINCIPLE ON

(458) **MUSCULAR FATIGUE.** (*Action du principe actif surrénal sur la fatigue musculaire.*) PANELLA, *Arch. ital. de Biol.*, T. 48 f. 3, 1908, p. 431.

THIS question, which is of fundamental interest as regards the internal secretion of the suprarenal gland, has given rise to evident differences of opinion on the part of many writers who have studied the problem. In order to solve it, Panella has gone very carefully into the matter, experimenting not only on heterothermic, but also on homothermic animals. These experiments are described in full detail, and the author sums up his results in the following general conclusions:—

1. The functional activity of a striated muscle in heterothermic animals is markedly strengthened by the supra-renal principle conveyed to the muscle by means of the circulation. Under the influence of the myosthenine (which the author considers the most suitable name for this product) the muscle can act rhythmically for a much longer time than under ordinary conditions.

2. Myosthenine, administered to the frog or the toad in an advanced period of fatigue, produces a recovery, marked, though not complete, of the functional activity of the muscle.

3. When the myosthenine is injected after the muscle has

become, as a result of the conditions under which it is acting absolutely incapable of contracting, it has no effect upon the muscular activity.

4. The same effects are also obtained in the muscles of homothermic animals (guinea pigs, rabbits), on condition, however, that by means of special treatment (section of the bulb, deep narcosis) they are placed in some degree under the respiratory, circulatory and thermogenetic conditions of heterothermic animals.

5. In homothermic animals it has not been possible to obtain the effects of the myosthenine upon muscular fatigue when it was injected into animals in which respiration, circulation and the production of heat differed somewhat from the normal. This is probably to be attributed to a profound modification which takes place in this substance when in contact with the blood. In fact:

6. The myosthenine loses its effect upon the muscles of the frog or toad when it has, for a comparatively short time, been in contact with the fresh arterial blood of the dog or the rabbit.

7. In frogs and hibernating toads, the action of the myosthenine is produced, if at all, only after a very considerable time. To obtain the effect of myosthenine as in warm-blooded animals, they need only be heated by keeping them in a temperature above 20° C. The cause of the difference between warm-blooded and hibernating animals is probably that in the former absorption is more rapid, and very much slower in the latter.

A. N. BRUCE.

PSYCHOLOGY.

**THE VARIATION OF THE ARTICULATORY CAPACITY FOR
(459) DIFFERENT CONSONANTAL SOUNDS IN SCHOOL
CHILDREN.** ERNEST JONES, *Inter. Arch. f. Schulhyg.*, Bd.
5, Ht. 2, S. 137-157.

THIS is a detailed investigation of (1) the order of frequency with which defects are shewn in relation to different sounds, and (2) the influence that the position of the sound in the word has on the facility with which it is enunciated; the two sexes are compared in respect to these questions. Amongst the conclusions appeared the following findings:—Th (especially in initial and final positions), Ng (especially final), The (especially final), and V (especially intermediate) were the sounds shewing greatest difficulty of enunciation. On the whole the girls excelled decidedly, most with (1) Th (especially final Them and Thed), Th (especially intermediate Thr and Thu), and in all the compounds of these two sounds, (2) in all sibilant sounds, and in nearly all their compounds. The boys excelled most with Ng and Zm. The influence of the position of the sound in the test word was

greatest in regard to final Ng, final The, intermediate V, initial Shr, which were all much harder to enunciate than the same sound in other positions. Eight tables accompany the article.

AUTHOR'S ABSTRACT.

THE IDEALS OF CHILDREN. (*Les Idéals d'Enfants.*) M. J. (460) VARENDONCK, *Arc. de Psy.*, July 1908, p. 365.

THIS paper describes a research into the ideals of children, the material being the answers of 745 school children to the question, What person whom you know, either through study or conversation, would you like to resemble? Several specimen answers are quoted. Five diagrams are given showing how choice is affected by age and sex.

MARGARET DRUMMOND.

CLASSIFICATION AND TABLE OF PSYCHOLOGICAL METHODS. (461) (*Classification et Plan des Methodes Psychologiques.*) ED. CLAPARÈDE, *Arc. de Psy.*, July 1908, p. 321.

THIS paper is an attempt to give a systematic classification of all the methods used in psychology. An elaborate scheme is expounded; four principal classes of methods are distinguished, and each of these is subdivided into the same six species. Under each of the twenty-four headings thus obtained illustrative examples are given.

As an introduction to this the author briefly sets forth and criticises the classifications recently advanced by Wundt, Ebbinghaus, Külpe, and others.

MARGARET DRUMMOND.

PATHOLOGY.

CONTRIBUTION TO THE PATHOLOGY OF FORCED MOVEMENTS IN CENTRAL LESIONS. (*Beitrag zur Pathologie der Zwangsbewegungen bei zentralen Herderkrankungen.*) MURATOW, *Monatsschr. f. Psychiat. u. Neur.*, Bd. 23, S. 510.

THE author first describes the case of a man aged 54, who four years previously had been attacked by a progressive right-sided hemiparesis. Following this came athetosis and hemi-hypoesthesia on the same side. At the autopsy was found an old gumma in the lateral nucleus of the optic thalamus.

The author ascribes choreic and athetotic movement to implica-

tion of the cerebello-thalamic tract at any point, and gives a differential diagnosis between affections of various sections of the tract, as also between cortical and thalamic lesions. He maintains that there is no sensory tract in the internal capsule.

ERNEST JONES.

CLINICAL NEUROLOGY.

ON THE PATHOGENESIS OF OPTIC ATROPHY AND OF THE
(463) SO-CALLED TOWER-SHAPED SKULL. (*Zur Pathogenese*
der opticus-atrophie und des sogenannten Turmschädels.)
 MELTZER (Chemnitz), *Neurolog. Centralbl.*, June 16, 1908,
 p. 562.

IN this paper Dr Meltzer draws attention to the frequency of peculiarly shaped heads in institutions for the blind, and in a series of 20 cases he points out the association between optic atrophy and a shape of head to which the Germans give the suggestive name of "Turmschädel" or tower-shaped. The Turmschädel is symmetrical, the top of the cranium rises high above the ears, and the apex of the tower is of very small dimensions. Of the 20 cases 3 were scaphocephalic, 5 were sphenoccephalic, and 12 oxycephalic, and most instructive sketches are given illustrating these peculiarities. In tabular fashion various points of interest are noted. Among these we may mention that 1 case had the typical shape of head at birth, while 12 cases had a less marked Turmschädel at birth, and the blindness came on between the first and sixth years of life, and at the same period the Turmschädel became more marked. The remaining 7 cases acquired the peculiar shape of head *after* becoming blind, and at ages varying from three months to three years of age. Nineteen out of the 20 cases had nystagmus, and 18 showed definite ex-ophthalmos. Nineteen had divergent and 1 convergent strabismus. Under etiology meningitis serosa was ascertained to be present in 14 cases, and 6 were the result of injury, while 17 out of the 20 were more or less definitely rachitic.

Dr Meltzer believes that meningitis serosa is the cause in most cases, producing a degree of hydrocephalus sufficient to exert destructive pressure on the optic, and in some cases also on the olfactory nerves. The ventricles are the site of the affection. In 13 of his cases the condition had commenced either before or at birth, while in the remaining 7 cases both the change in the shape of the head and the optic atrophy developed later, but within the first three years of life. The author believes that the peculiar shape of the head is due to hydrocephalic pressure on bones

which have a tendency to rickets. After ossification of the bones and union of the sutures the developing brain aids reabsorption of the fluid, while it may induce rarefaction of the vault and some deformity at the base of the skull. The pressure in all cases is responsible for the optic atrophy, and the interference also with the olfactory nerves in some instances.

The author suggests that lumbar puncture should be tried when the communication with the ventricular system is intact, and when it is not, the lateral ventricles should be punctured or trephining performed. Whichever operation is carried out, it should anticipate the complete loss of sight. R. A. FLEMING.

THE PATHOGENESIS OF TABES DORSALIS. TOM WILLIAMS, (464) *Amer. Journ. Med. Sc.*, Aug. 1908, p. 206.

CONTROVERSY as to the pathogeny of tabes has not ceased since Duchenne surmised its sympathetic origin, and Charcot later supposed it to be a posterior column dystrophy, similar to that of Friedreich's ataxia. His great authority prevented due attention to the researches of Obersteiner and his followers until the memoir of Redlich appeared in 1896. The views as to the syphilitic etiology of tabes drew greater attention to the work of Nageotte, who in 1894 had indicated the constancy of lesions of the radicular nerve at the point where it receives its meningeal sheaths. These lesions correspond to one or other of the stages of the granulomatous process, varying as they do from simple round cell infiltration to granuloma, and even breaking down with formation of cavities. They are due to primary chronic meningitis, evidenced by the lymphocytosis found by spinal puncture during life and post-mortem when skilfully looked for, although the tendency of the process to resolution and fibrous-tissue formation leaves only a slight thickening in the membrane, already fibrous by nature.

The changes in the cord are consecutive to this. That this is so is proved by similar changes of the posterior column after disease or experimental section of individual roots, and by the changes occurring in the radicular nerves as the result of increased pressure due to the growth of cerebral tumours.

The noxa falls unequally upon the various root fasciculi, and this corresponds to the disparate nature of the sensory troubles, which are not confined so strictly as was formerly supposed to the fibres which subserve the sense of attitude and of muscular movement; for it is now definitely known that cutaneous sensibility is always involved more or less, though probably later in the disease.

The superficial lightning pains described by Gowers and the psychometric analysis of the sensibility of tabetics by Vaschide

are an index of this; while the researches of Head enable us to explain the modifications in terms of deep, protopathic, and epicritic sensibility. The fibres subserving the life of internal relation may differ morphologically from those subserving external relation, as contended by Pierre Bonnier, with particular reference to the VIII. nerve, where the cochlear portion, whose function concerns the outside world, is affected only rarely, while the vestibular portion, concerned with intrinsic relationships, is involved very commonly indeed in the tabetic process.

However this may be, it is certain that impaired sense of attitude is always accompanied by impairment of the deep pain sense, and of perception of the vibrations of the tuning-fork by the bones, and as these sensory impressions are conveyed in the same peripheral path, while they are separated within the cord, clinical evidence is in entire harmony with the pathogenetic theory advanced by Nageotte. The data furnished by the optic nerve symptoms are similarly best explained by a meningeal affection, involving in this case not a posterior root but a homologue of an intra-spinal path.

The tabetic symptoms referable to the sympathetic do not differ from those produced by experimental section of the spinal roots, nor from those in syringomyelia, which, however, attacks the cell bodies in the intermedio-lateral columns. Charcot's negation of changes in the sympathetic is effectively disproved by the researches of Roux, who found the medullated fibres markedly decreased in tabetics.

The anterior roots are not unaffected, but the relative absence of serious myopathies early in the disease is accounted for by the rapid regeneration of the fibres. This is shown by the "*terminaisons en croissance*" exhibited in Nageotte's preparations and by the results of section experiments. The regeneration of the posterior root fibres extends only to Redlich's ring, at which they lose the neurilemma sheath.

Finally, evanescent lymphocytosis and reflex iridoplegia, the two most characteristic signs of tabes, are found in many cases of syphilis without other tabetic symptoms; indeed, both sometimes occur in the secondary stage, the former in as many as 40 per cent. of cases.

The contention of Babinski and Nageotte is therefore accepted that a chronic syphilitic meningitis is responsible for what has been called tabes dorsalis, and that it was formerly disregarded on account of the tendency to the occurrence of resolution and fibrosis of the lesions.

The practical application of this conclusion is of the greatest importance in the treatment of the disease. Cases taken early may be completely arrested, and in all cases the active manifesta-

tions may be resolved if adequately treated before the destruction of the noble elements has occurred, though naturally the residues of former exacerbations cannot be removed.

AUTHOR'S ABSTRACT.

TABES WITHOUT LIGHTNING PAINS. (*Le tabes sans douleurs* (465) *fulgurantes.*) J. ABADIE et NOGUE, *Jour. de méd. de Bordeaux*, 1908, p. 37.

THE writers examined 400 cases of tabes in Pitres's clinique, and found that 16, or 4 per cent., had never suffered from lightning pains. Only 2 of these patients had suffered from tabes for more than ten years, and only 4 for more than five and less than ten years. In most of the cases the other symptoms of tabes were ill-marked. Some of them presented other sensory disturbances, *e.g.* numbness, stiffness, and lassitude, but these symptoms were of slight intensity.

J. D. ROLLESTON.

CO-EXISTENCE OF TABES IN THE MOTHER AND ACTIVE (466) **SYPHILIS IN THE INFANT.** (*Coexistence du tabes chez une malade et de syphilis en evolution chez son enfant nouveau-né.*) H. DUFOUR et COTTENOT, *Bull. et mém. de la Soc. méd. des Hôp. de Paris*, 1908, p. 953.

A CHILD, aged one month, was admitted to hospital with typical congenital syphilis. Death took place four days later. The mother gave the following history:—Five years previously the husband had been under treatment, the nature of which he had concealed from her. The following year she had lost much of her hair, and subsequently had given birth to a premature still-born child. During the last two months she had suffered from lightning pains. She had had no treatment. Examination showed that the right knee-jerk was lost and the left was very weak. The right ankle-jerk was very weak, and the left was lost. The pupils were unequal. Reaction to light was very sluggish in the left, and almost absent on the right. Lumbar puncture showed abundant lymphocytosis. The case proves that, contrary to the opinion expressed by many authorities, tabetics should undergo treatment, since they are capable of transmitting active syphilitic lesions to their offspring.

J. D. ROLLESTON.

THE ETIOLOGICAL TREATMENT OF TABES. (*Le traitement (467) étiologique du tabes.*) MILIAN, *Progrès médical*, 1908, p. 301.

Of all parasymphilitic affections tabes seems to benefit most from mercurial treatment. After mentioning the preventive action of mercury, Milian states that complete cure of tabes by this drug is not very rare, while arrest of the disease is still more frequent. Certain symptoms, such as lightning pains, may be caused to disappear entirely, but the action of mercury in this respect is not constant, for it apparently aggravates the pains of tabetics in whom ataxia is present, while it relieves those in whom the symptoms are ill-developed (*tabétiques frustes*).

J. D. ROLLESTON.

A CASE OF ANTERIOR POLIOMYELITIS OF SPECIFIC ORIGIN. (468) (*Cas de Poliomyélite antérieure d'origine spécifique.*) A. VAN GEHUCHTEN, *Le Névrose*, Juin 10, 1908, p. 331.

THE case was that of a man of 32, who, twelve years after a syphilitic infection, developed a muscular paresis and atrophy, leading in about nine months to complete disappearance of the affected muscles. The left upper arm was first affected; several months later, the right. The order of involvement was the same in both, starting in the muscles of the shoulder girdle (deltoid, supra- and infra-spinatus, and sub-scapularis (?)), then spreading to the biceps and brachialis anticus, and lastly, the muscles on the outer aspect of the front of the forearm. The triceps, muscles on inner side of and behind the forearm, and muscles of the hands, were spared entirely. There were no sensory disturbances, the reflexes and motility of the lower limbs were unaffected, there was no E.R.D. in the affected muscles, simply a progressive loss of contractility. Clinically, an atrophy of the Aran-Duchenne type, the author considers it to be probably an anterior poliomyelitis, the result of syphilitic vascular disease, and arrested by vigorous mercurial treatment. Apparently the 5th and 6th cervical segments were involved. Some consideration follows of the relationship between the segmentary nuclei of the cervical enlargement and the anterior nerve roots.

J. H. HARVEY PIRIE.

ANALYSIS OF SEVENTY-SIX CASES OF POLIOMYELITIS (469) **ANTERIOR ACUTA.** BYROM BRAMWELL, *Clinical Studies*, July 1, 1908, p. 371.

THE author analyses seventy-six cases of this disease according to sex, age, initial symptoms, period of the disease at which the paralysis was first noticed, distribution of the initial paralysis and of the residual paralysis.

EDWIN BRAMWELL.

**DISEASE OF THE PRIMARY MOTOR NEURONES CAUSING
(470) THE CLINICAL PICTURE OF ACUTE ANTERIOR POLIO-
MYELITIS: THE RESULT OF POISONING BY CYANIDE
OF POTASSIUM. JOSEPH COLLINS and HARRISON S. MART-
LAND, *Journ. Nerv. and Ment. Dis.*, July 1908, p. 417.**

IN a fairly careful search of the literature, the authors have been unable to find any cases of neuritis or poliomyelitis due to cyanide of potassium. It is unnecessary to describe in detail the symptoms of the case here recorded, for the reporters of the case say that it is indistinguishable from an anterior poliomyelitis. The patient was a silver polisher in a hotel. His work was to drop the silver into a solution of cyanide of potassium and then to dry it. His hands and arms had taken on a deep red brown colour, were frequently distressingly itchy, and his finger nails were quite black. His illness began with severe diarrhoea. The following day he complained of severe headache and pain and stiffness in the back of the neck. He was mildly delirious, and for a few days had some neck retraction. On the fifth or sixth day his arms and legs were so weak he could scarcely lift them. On the eighth day he had retention of urine. The patient made a slow recovery, and at the time of writing was still improving.

The authors have made some experiments on rabbits, administering small but poisonous doses of potassium cyanide. Paralysis of the hind limbs, with loss of the reflexes, convulsions, and incontinence, resulted. Central chromatolysis with nuclear eccentricity, vacuolisation, and shrinkage, with almost complete solution of the cell, was observed in the anterior horns from the lower cervical region to the upper lumbar region, varying in degree, but most marked in the mid-dorsal part of the cord. The changes observed in the anterior horn cells were, in the opinion of the authors, due to an extension of a peripheral multiple neuritis, *i.e.* of an axone degeneration so severe that it ultimately affected the cell itself.

EDWIN BRAMWELL.

**SYRINGOMYELIA WITH SYRINGOBULBIA. JOHN H. W. RHEIN,
(471) *Journ. Med. Research*, March 1908.**

THIS article consists of the clinical report of Syringomyelia with Syringobulbia in a woman aged 59 with a sixteen years' history, of a careful pathological examination, of a full reference to the literature bearing on the case, of descriptive plates, and of a discussion of the case observations being put forward which warrant

the conclusion that syphilitic disease of the cord and meninges in syringomyelia is probably more than a coincidence.

The lesion in syringobulbia does not, except in one reported case (Spiller), extend beyond the lower part of pons, and yet cranial nerves above nucleus of facial nerve are frequently involved in cases of syringobulbia. The author states that symptoms in distribution of nerves above the seventh may be due to pressure upon them by a co-existing leptomeningitis. In case under consideration there was peripheral degeneration from pressure of intense leptomeningitis found in third nerve, in fourth nerve, in fifth nerve, in right ninth and tenth nerves, in both eighth nerves, in right seventh and both optic nerves.

The distribution of the cavity and resulting degeneration is very fully described, other clinical features of case being left hemiplegia, ataxia, atrophy of hand muscles and of right half of tongue. Characteristic sensory disturbances were observed.

W. KELMAN MACDONALD.

SPINAL SPRAIN: Its Complications and Consequences, with Report (472) of Cases. P. LE BRETON (Buffalo), *Jour. Am. Med. Assn.*, May 23, 1908.

THE object of the author is to review the general subject of spinal sprains from a clinical and practical standpoint, emphasizing the importance and frequent necessity of fixation.

The clinical groups are: (1) sprain; (2) sprain followed by neurotic symptoms; and (3) sprain accompanied by spinal cord symptoms. Injuries are more commonly indirect than direct; the site is most often the lumbar dorsal region.

The pathological changes are those of a sprain, the ligaments about the spinal column are torn, muscle and fibrous tissue lacerated, exudation of blood and serum into the tissues, and often the formation of a blood clot in the spinal canal which presses on the cord. As a direct result there may be temporary loss of function, meningitis or pachymeningitis, but the commonest condition is a subsequent zonal paralysis or mild transverse myelitis, from which recovery is partial or complete.

The diagnosis is based upon the history of injury, change of posture, stiff gait, drawn facies, contour of spine, tenderness, and limitation of spinal motion (due to pain or spasm).

The outlook is better if the diagnosis is made early and treatment is prompt, and the author strongly approves of some sort of mechanical support properly adjusted to the needs of the individual case, and applied early.

His paper includes the case histories of ten patients whose symptoms varied from those of simple sprain to neurotic and to spinal cord symptoms.

C. H. HOLMES.

THE THERAPEUTICAL VALUE OF LUMBAR PUNCTURE IN (473) THE MENINGEAL FORMS OF TYPHOID FEVER IN CHILDREN. (La ponction lombaire dans les formes meningées de la fièvre typhoïde chez l'enfant. Son utilité thérapeutique.) ROCAZ et CARLES, *Bull. et mém. de la Soc. de méd. et de chir. de Bordeaux*, 1907, p. 453, and *Gaz. hebd. des sciences méd. de Bordeaux*, 1908, p. 39.

TYPHOID fever in children is often accompanied by meningeal symptoms, which may be slight and transitory, but are sometimes more pronounced and persistent. Four chief forms are described. 1. In the second or third week the "meningeal tripod" of headache, vomiting, and constipation appears, followed by cutaneous hyperæsthesia, irregular pulse and respiration, and ocular troubles. The symptoms either disappear in a few days or end in death. 2. The complete picture of cerebro-spinal meningitis. In addition to the symptoms present in the first form, Kernig's sign and stiffness of the neck and vertebral column, are present, and the hands and feet assume the tetany position. This is the form most frequently met with. 3. Typhoid fever at the onset may closely resemble tuberculous meningitis. 4. A form peculiar to nurslings characterised by subintractant convulsions which prove rapidly fatal. Lumbar puncture may show (a) pus containing typhoid bacilli either pure or associated with staphylococci; (b) a turbid or even transparent fluid in which typhoid bacilli and other organisms are found on centrifugalisation; (c) a clear fluid containing abnormal cellular elements, and showing as a rule abundant lymphocytosis; (d) a liquid of normal composition, but escaping in a jet. In the first two cases there is a direct microbial infection of the meninges, and in the last two probably a meningeal irritation due to microbial toxins. Lumbar puncture is of value in that it removes the microbes and their toxins, diminishes the compression of the nerve centres, and causes disappearance of the symptoms. Eight illustrative cases, hitherto unpublished, are recorded. All recovered.

J. D. ROLLESTON.

THE SYMPTOM-COMPLEX OF OCCLUSION OF THE POSTERIOR (474) INFERIOR CEREBELLAR ARTERY: TWO CASES WITH NECROPSY. WILLIAM G. SPILLER, *Journ. Nerv. and Ment. Dis.*, June 1908, Vol. xxxv., No. 6.

THE symptom-complex of occlusion of the posterior inferior cerebellar artery is usually sharply defined, although it may be difficult to exclude implication of the vertebral artery. The onset is usually

sudden and without disturbance of consciousness. The limbs are not paralysed, or at most are paretic on the side opposite the lesion, and the paresis is not persistent. Pain and temperature sensations are diminished or lost in the limbs of the side opposite the lesion and in the whole or a part of the fifth nerve distribution on the side of the lesion, occasionally also in the face on the side opposite the lesion. Spontaneous pain or paræsthesia may be felt in the area of disturbed objective sensation. Tactile sensation and sense of position are usually intact. Ataxia may be present in the limbs on the side of the lesion, with a tendency to fall toward the side of the lesion. Paralysis of the muscles of deglutition, of the soft palate and larynx occurs on the side of the lesion, with smallness of pupil, retraction of eyeball and narrowing of palpebral fissure (sympathetic paralysis) on the side of the lesion. Hiccoughing and vomiting may be obstinate, and the pulse may be rapid from paralysis of the vagus. The deep reflexes usually are diminished or lost, but may be exaggerated. Headache may be intense. These are the principal symptoms, but there may be others—nystagmus, vertigo, disturbance of micturition, paresis of the tongue, of the seventh nerve distribution and external rectus, and impairment of taste on the side of the lesion, etc., depending on the extent of the thrombus. The author has found sixteen reported cases in which necropsy was obtained; there are others purely clinical. The right posterior inferior cerebellar artery is sometimes absent, and the lesion is more commonly left-sided. The occlusion affects the lateral and posterior part of the medulla oblongata without extending to the periphery and without producing lesions of the cerebellum, as the anastomosis in the latter is usually sufficient to prevent softening. The symptoms are explicable by the anatomy of the part affected. The author reports two cases with necropsy in which the symptoms and lesions were typical, although in the second case the lesion extended a little further anteriorly than in some of the reported cases. AUTHOR'S ABSTRACT.

A CASE OF CHRONIC MIDDLE EAR SUPPURATION COMPLICATED BY TUMOUR OF THE PONS (GLIOSARCOMA).

(Ein Fall von chronischer Mittelohreiterung kompliziert mit Ponstumor (Gliosarcom).) W. KÜSTNER, *Arch. f. Ohrenhklde.*, H. 3, p. 181, 1908.

THE patient, a woman æt. 23, had suffered from running ears for six years. Four months and two months before admission she had had attacks of giddiness coming on after dancing, and on the second occasion she also noticed weakness of the right leg. Three weeks later she had an attack of vomiting, and this recurred every

morning. She also suffered from severe headaches, her condition became so rapidly worse that she was admitted to hospital. On examination, the sixth nerve on the left side was found to be paralysed, there was some redness of the left disc, the face was paralysed on the left side, and taste (anterior two-thirds of tongue) and smell were also impaired. Nystagmus appeared on deviation of the eyes to the right. There was loss of power in the right arm and leg, and the sense of touch was impaired. The deep reflexes were exaggerated and the plantar reflex showed dorsiflexion.

A radical mastoid operation was performed on the left side, and the temporal lobe was also explored, with a negative result. Two months later the patient died. At the section a glioma was found in the left half of the pons.

W. G. PORTER.

THE INDICATIONS FOR OPERATIVE TREATMENT IN CHRONIC (476) INFLAMMATIONS OF THE FRONTAL SINUS. (Ueber Indikationen zur operativen Behandlung bei der chronischen Stirnhöhlenentzündung.) M. HAJEK, *Wien. med. Wchnsch.*, Bd. 58, H. 26, S. 1466.

THE author asserts that the indications for operative treatment in inflammation of the frontal sinus have not been sufficiently discussed, for the views of various authorities are widely at variance. In acute inflammation opinions cannot differ widely. Most cases heal without operation, a number with endonasal treatment, and those alone require a radical operation where the bone is also diseased.

Chronic Inflammations.—Apart from those cases in which there is diseased bone and evidence of intracranial complications, and where there is no question but that a radical operation is required, the majority of cases consist of those showing a clinical picture of nasal discharge accompanied by headaches. If treated by the usual endonasal methods these can be divided into three classes:

1. A few cases which entirely heal in a short time.
2. Many cases which are cured of headache but where the discharge continues.
3. Many cases in which the headaches are not cured and the discharge does not stop.

If we consider groups 2 and 3, the question arises—How long are we to wait before proceeding to the radical operation? Hajek is of the opinion that we can safely wait for months, especially if the pain is relieved, for the risk of intracranial complications is exaggerated. It should also be remembered that the radical operation is not unaccompanied with risk.

W. G. PORTER.

TUMOUR OF THE FRONTAL LOBES, WITH SYMPTOMS SIMULATING PARESIS. F. X. DERGUM, *Journ. Nerv. and Ment. Dis.*, July 1908, p. 438.

THE mental symptoms in this case "in a way suggested paresis." The patient's manner was very like that of a paretic. He did not realise that he was ill. He treated everything as a joke. His habits and conduct were greatly changed. He became indifferent as to his professional duties and unpunctual in his appointments. Headache, defective vision, more especially in the right eye, and due to a postneuritic atrophy, loss of smell, and some tremor of the tongue and lips were practically the only additional positive signs present. Post-mortem, an enormous encapsulated sarcoma was found involving both frontal lobes. EDWIN BRAMWELL.

HERPES ZOSTER AND MUMPS. (*Zona et oreillons.*) APERT, (478) *Médecine Moderne*, 1907, p. 210. (Soc. de Péd.)

A CHILD, aged 8 years, developed herpes zoster forty-eight hours before the appearance of mumps. Lumbar puncture gave issue to a clear fluid which contained only a few lymphocytes. There was, therefore, no meningeal reaction. J. D. ROLLESTON.

DYSPRAXIA IN LEFT HEMIPLEGIA. (*Dyspraxie bei linksseitiger Hemiplegie.*) HILDEBRANDT, *Neurol. Centralbl.*, Juni 16, 1908, p. 576.

A CASE of left hemiplegia in which there was dyspraxia in the right hand. The patient was left handed, but had practised movements with the right hand. This observation, in the author's opinion, supports the view advanced by Liepman that the condition is intimately connected with right-handedness.

EDWIN BRAMWELL.

URÆMIC HEMIPLEGIA AND APHASIA. (*Hémiplégie droite et aphasie urémiques.*) R. DUPÉRIÉ, *Jour. de méd. de Bordeaux*, 1908, p. 119.

A FEMALE, aged 73 years, who was supposed to have senile myocarditis, suffered from syncopal attacks in which considerable increase in the volume of the liver, oliguria, and albuminuria were

observed. After appropriate treatment the hepatic dulness diminished, and the urine became normal in twenty-four hours. An unusually severe syncopal attack subsequently developed, in which right hemiplegia and aphasia occurred. Oliguria and albuminuria were pronounced. Death, preceded by coma and Cheyne-Stokes breathing, took place five days later. The diagnosis of cerebral embolism was made, but at the autopsy no trace of endocarditis or cardiac thrombosis was found. The kidneys showed the lesions of chronic nephritis. There was no cerebral oedema, and no recent macroscopical lesions of the left hemisphere could be found, but only a small and old-standing erosion of the inferior parietal convolution. Dupérié concludes that the nerve centres were intoxicated by the accumulation of urinary poisons, and attributes their localisation to the presence of the pre-existing lesion.

J. D. ROLLESTON.

SPLITTING OFF OF THE COLOUR-SENSE. (*Ueber Abspaltung (481) der Farbensinnes.*) LEWANDOWSKY, *Monatsschr. f. Psychiat. u. Neur.*, Bd. 23, S. 488-510.

THE essential points of the case here carefully described and analysed are as follows:—A man of 50 suffered from sensory aphasia (of the typical complete Wernicke form), due probably to embolism. As this recovered certain remarkable symptoms were left behind. Thus, though the patient could distinguish all shades of colour, and so had no colour-blindness, he could not (1) name the colour of any object shewn or mentioned to him, (2) pick out named colours, (3) pick out colours of objects known to him. The condition was obviously not a part of any agnosia, for the patient recognised the use, meaning, and name of all objects.

Wilbrand described twenty-four years ago a condition that he called amnesic colour-blindness, in which the patient was unable to name the colours of objects, though he had no true colour-blindness. Many similar cases have been described, but Lewandowsky doubts the existence of any such condition, inasmuch as the presence of other defects above-mentioned was never inquired into in the cases recorded. He therefore refuses Wilbrand's explanation of them as being due to a separation between the colour-sense centre and the speech centre (a form of transcortical motor aphasia), and with very ingenious and close reasoning, supported by various forms of testing, traces the defect to an inability to associate the colour and form of an object. The patient could not conceive the colour or name of red, for instance, because he could not associate the colour with objects, blood, cherries, etc., that it was previously associated with.

ERNEST JONES.

THE SIGNIFICANCE OF PHRICTOPATHIC SENSATION. ERNEST
(482) JONES, *Journ. Nerv. and Ment. Dis.*, July, Vol. xxxv., p. 427.

THIS article contains, first, a description, and then an analysis of this syndrome that is met with during the recovery from certain forms of hysterical anæsthesia. The six characteristic features of it are as follows:—(1) Abnormal persistence; instead of the sensation ceasing to be experienced immediately the cutaneous stimulus is withdrawn, as in the normal, it here persists in unabated intensity for a variable time, even up to sixty seconds. (2) Delayed reaction time; an interval of a few seconds elapses before the stimulus is apprehended. (3) Non-perception when a more normal sensation is present; the stimulus is not appreciated if a normal part is simultaneously stimulated, and the abnormally persisting sensation is instantly abolished if a normal part is subsequently stimulated—this even if there is no trace of hypæsthesia in the abnormal part. (4) Tendency to immediate motor response; stimulation is followed by an irresistible sudden jerk. (5) Disagreeable quality; the quality is that of a disagreeable radiating shudder, which may be intensely unpleasant. (6) Impairment of the sense of personal ownership of the part; this sense may be diminished or totally abolished.

The fourth and fifth of these features were erroneously described as haphalgæsia seventeen years ago by Pitres, who was under the impression that they could be induced only by the application of certain precious metals.

The explanation given of the syndrome is as follows:—*Æsthetic sensibilities*—(a) common, (b) *coenæsthetic*—can be abrogated by either physical or mental disease; *autosomatognostic memories* of a given part of the body only by mental. Hence we have two forms of hysterical anæsthesia: (1) the common one in which only the various sensibilities are lost, and (2) a less common one in which the memories concerning the part are also lost. In the latter case, when recovery occurs, both groups of mental processes are usually recovered together. If the former group is recovered before the latter, phrictopathic sensation occurs. Thus the intensity of the features of the phrictopathic sensation is an accurate measure of the extent of the cleavage between the *æsthetic sensibilities* and the *autosomatognostic memories*.

AUTHOR'S ABSTRACT.

A CASE OF RECURRENT AUTOHYPNOTIC SLEEP. BERNARD
(483) OETTINGER, *Journ. Nerv. and Ment. Dis.*, March 1908.

AFTER a prolonged sleep of thirty-five days, patient woke up mute, and on day after was apparently deaf, the deaf mutism lasting

for four months and nineteen days. An actual delirium was later developed after a temporary state of hypomania.

The author advocates the extension of the scope of psychotherapy by systematic use of disagreeable as well as pleasing emotions, especially when avenues of sight and hearing are not available to conversational suggestion. In reported case patient was aroused by frequent cool baths after apomorphine (in emetic doses) and strong induced faradic current had failed.

W. KELMAN MACDONALD.

COLONY AND BROMIDE TREATMENT OF EPILEPSY. A. J. (484) M'CALLUM, *Brit. Med. Journ.*, March 14, 1908.

AFTER giving statistics of results obtained at the Epileptic School for Boys at Stamthwaite, Westmorland, the author discusses the cause of epilepsy, throwing aside the "abnormal state of chemical nutrition" adduced by Gowers, and maintaining that epilepsy becomes a reflex act in its origin and a cerebral vice in its fuller development and perpetuation.

Bromide of potassium is used in the treatment, the dose being the amount necessary to control the fits—40 grs. sometimes, 100 grs. often, and even 300 grs. per diem.

Method is to give 20 grs. night and morning, and increase by 10 grs. per day as often as fits recur. Diet is summed up in three meals a day, everything fresh, everything limited, flesh never oftener than once a day, and three times a week is ample.

W. KELMAN MACDONALD.

THE MENTAL STATE IN CHOREA AND CHOREIFORM (485) AFFECTIONS. CHARLES W. BURR, *Journ. Nerv. and Ment. Dis.*, June 1908.

FOR the better study of this subject cases are divided, as far as mental symptoms are concerned, into four groups:—First, peevishness, fretfulness, and selfishness; second, transitory hallucinations; third, delirium; and fourth, stupor or acute dementia. No mental symptoms are regarded as pathognomonic of chorea, and the author denies existence of a disease "chorea insaniens," but regards chorea in childhood as an indication of inherent nervous instability requiring careful teaching of child in self-control. Numerous illustrated cases are given.

W. KELMAN MACDONALD.

**BLOOD PRESSURE IN NEURASTHENIC STATES AND THE
(486) EFFECT OF CERTAIN FORMS OF TREATMENT THEREON.**

ERIC D. MACNAMARA, *Lancet*, July 18, 1908, p. 151.

THE author summarises his conclusions as follows :—

1. In many cases of neurasthenia there is an alteration of the level of the blood pressure from the normal, the level being sometimes higher and sometimes lower than normal.

2. Patients who show such alterations and who undergo certain sorts of treatment manifest, in some cases, at the end of a course of treatment a level of pressure different from that which existed before treatment began. If the pressure at the beginning be abnormally high it will probably descend, while if it be abnormally low it will probably ascend.

3. The number of patients whose blood pressure is different at the end of their course of treatment from that which it was before treatment began is greater among those without a family history of nervous instability than among those with such a history.

4. In a very large majority of cases the application of high-frequency currents produces a lowering of blood pressure, while the static bath (plus charge) and massage produce a raising of pressure. The discharging of a patient charged with static electricity results as often in a rise as in a fall of blood pressure. The application of the faradic current tends rather to lower than to raise the blood pressure, and the galvanic current cannot be said to effect much change in either direction.

5. It is difficult to institute treatment with any confidence that there will be at the end of the course such an alteration as might perhaps have been expected from a knowledge of the alteration that is likely to follow each application of the therapeutic agent employed, though we may look for a change of level when the level was at the commencement abnormal to one that is nearer the normal.

6. There is no evidence that the differences of variation of level before and after treatment which may sometimes be noted can be correlated with improvement of health.

AUTHOR'S ABSTRACT.

AUTO-SUGGESTION IN NEURASTHENIA. (L'Autosuggestion
(487) chez les neurasthéniques.) HARTENBERG, *Rev. de Méd.*, Juin,
p. 561.

THE author briefly mentions ten cases in support of his opinion that auto-suggestion plays no part whatever in true neurasthenia, and that when it is present some other condition exists, such as hysteria, disequilibrium, neuropathism, etc.

ERNEST JONES.

INCOMPLETE MYXŒDEMA. A. R. ELLIOTT (Chicago), *Jour. Am. (488) Med. Assn.*, May 3, 1908.

THE object of the paper is to draw attention to a condition of hypothyroidia or insufficiency of the gland secretion, which is probably an incipient or early stage of myxœdema. The symptoms are so slight that they are often not recognized, or are mistaken altogether. They are the same as in fully-developed myxœdema, but less intense, and not typically grouped. They disappear under thyroid feeding, as do those of the fully-developed disease. Most of these cases of hypothyroidia are encountered in women about the time of the climacterum, and are often attributed to the menopause—the predominance of all myxœdematous states in the female over the male is in the ratio of 7 to 1.

Some of the most important symptoms by which this condition is recognized are: transient œdemas, scaly skin, joint pains, neuralgias, parasthesias, extreme susceptibility to cold, yellow tinging of the skin, disposition to obesity, mental dulness; the thyroid may or may not be diminished in size, sometimes it is even enlarged.

Elliott reports two of his own cases which were diagnosed early, given prompt treatment by thyroid feeding, and made good recoveries.

C. H. HOLMES.

TO WHAT EXTENT IS MIGRAINE AMENABLE TO TREATMENT (489) OF THE EYES? E. M. ALGER (New York), *N.Y. Med. Jour.*, June 6, 1908.

AFTER a brief discussion of the symptoms and course of the disease, with an attempt to define clearly his position as to what should warrant a diagnosis of "Migraine," the author turns to the importance of eye strain as a causative factor in its development. He states that three per cent. of cases develop between the ages of 5 and 10 years, or at the time when school life first puts a strain on the eyes; in old age, when accommodative power has practically disappeared, migraine attacks almost never occur; abuse of the eyes often precipitates an attack in apparently healthy people. The three ocular disturbances which are most concerned in the development of this condition are: (1) over-use of the ciliary muscle in accommodation, such as occurs in hyperopia and astigmatism; (2) conditions in which binocular vision is impossible without undue strain of the extrinsic ocular muscles; (3) cerebral fatigue that comes from the continual interpretation of distorted retinal images, such as are present in astigmatism and aniso-

metropia. Ten cases are cited in which, after careful study, it was decided that ocular disturbances were the cause of the trouble. Attempts were made to correct these, and improvement with ultimate recovery followed.

C. H. HOLMES.

HEART FAILURE IN EXOPHTHALMIC GOITRE. (*L'asystolie (490) mortelle dans la maladie de Basedow.*) G. MOURIQUAND et LÉON BOUCHUT, *Semaine méd.*, 8 Juillet 1908, p. 325.

A CRITICAL analysis of the literature upon this subject leads the authors to conclude that there is no convincing evidence of heart failure, induced simply by tachycardia, being ever the sole cause of death in exophthalmic goitre. In most of the cases which terminated by heart failure there was some antecedent cardiac disease, and the fatal issue was merely accelerated by the tachycardia. In a second group of cases there was some other concomitant disease, such as pleurisy or Bright's disease, but in these cases the heart would probably not have been regarded as healthy had the myocardium been examined microscopically. Finally, there are cases where at first sight there did not appear to be any other cause for the heart failure than the tachycardia. These cases, however, have usually run a rapid and progressive course, and the morbid changes both in the thyroid and in the heart can be referred to some recent and general infective disease, and most commonly to a rheumatic infection.

W. F. RITCHIE.

THE X-RAY TREATMENT OF EXOPHTHALMIC GOITRE. (491) C. THURSTAN HOLLAND, *Arch. of the Roentgen Ray*, July 1908.

THE writer publishes his results in a series of twenty cases. The method adopted was to expose either side of the neck alternately to X-rays for ten minutes two or three times a week. The most prominent results were an immediate drop in the pulse-rate in nearly all the cases, together with marked improvement in the muscular tremor and general nervousness. The gland itself sometimes diminished in size, but what was more noticeable was the fact that in the cases where the gland was hard and tense, after a very few exposures it became much softer. The exophthalmos did not materially alter. It was pointed out that there was a danger in these cases of producing myxœdema, and the suggestion was made that in future cases it would be wise to treat one side of the gland only.

The author is strongly in favour of this method of treatment, which, of course, can be combined with the usual medicinal methods.

AUTHOR'S ABSTRACT.

MENTHOL POISONING IN MAN. (Ueber Mentholvergiftung des (492) Menschen.) SCHWENKENBECHER, *Münch. med. Woch.*, 1908, No. 28, p. 1495.

It is known that the external application of menthol produces a cold sensation. The writer has shown that by stomach administration of 8 grammes of menthol there is induced, in addition to slight symptoms of general intoxication, a condition of local and remote temperature paræsthesia.

Thus a cold sensation is felt in the mouth, throat, and œsophagus immediately on swallowing the menthol, in the nose a little later, and in the anus during excretion of menthol in the fæces. The absence of this sensation in the stomach and abdomen agrees with the observation that the mucous membranes of the stomach and intestines possess no "cold sensitive" nerves.

There is also produced a hyperexcitability of the "cold sensitive" nerves in the skin, so that anything touched by the hand feels ice-cold, showing that menthol exerts a selective stimulating action on these nerves also when conveyed to them by the blood.

J. A. GUNN.

MULTIPLE RELAPSING GANGRENE OF THE ARMS AND (493) FOOT. AMPUTATION OF THE LEFT ARM. DISCUSSION ON THE NATURE OF THE GANGRENE. PATHOMIMESIS. (Escarres multiples et recidivantes depuis deux ans et demi aux deux bras et un pied. Amputation du bras gauche. Discussion sur la nature des escarres. Pathomimie.) DIEULAFOY, *Bull. de l'Acad. de méd.*, 1908, p. 635, and *Presse Médicale*, 1908, p. 369.

A WELL-EDUCATED man, aged 30, free from any alcoholic or nervous taint, consulted Professor Dieulafoy in April 1908 for a gangrenous affection of the arms of two and a half years' duration. During this period he had consulted numerous physicians and surgeons, who had respectively diagnosed neuritis, syphilitic or tuberculous ulceration, and hysterical trophic troubles, and subjected him to the most varied treatment without effect. A surgeon who diagnosed trophic ulceration following neuritis performed elongation of the nerves of the left brachial plexus. The pain in the arm then became intolerable, requiring injection of morphia day and night,

and there was a commencement of claw-hand in the ring and little finger. The surgeon now declared that amputation was the only means of avoiding the reproduction of the gangrene, and the operation was performed through the upper third of the upper arm on August 21, 1906. All went well until February 1907, when patches of gangrene began to appear again on the right arm. Elongation of the corresponding brachial plexus was recommended, but this time was refused. At his first visit to Dieulafoy the patient had no less than 98 lesions on his arms, and a few days later similar lesions began to appear on one foot. Dieulafoy found no evidence of syphilis, diabetes, tabes, or neuritis, and never having seen trophic troubles following hysteria (v. *Rev. of Neurol. and Psych.*, p. 426, 1908), he concluded that the lesions were the result of simulation and were due to the application of caustic potash. The accuracy of this diagnosis was admitted by the patient, in a dramatic scene, in which Dieulafoy told him that though up to that moment he had been acting under an irresistible impulsion, persistence in such conduct would stamp him as an impostor. Dieulafoy has adopted the term "pathomimie" (pathomimesis), supplied him by the well-known writer Paul Bourget, to designate the mental condition of such persons, who, in addition to the systematic fabrication of falsehoods (mythomania of Dupré), simulate diseases either with a fraudulent intent, or, as in the present case, without any such motive, but simply from a disinterested love of mystification.

J. D. ROLLESTON.

PSYCHIATRY.

PUPILLARY PHENOMENA IN THE INSANE. (Contribution à (494) l'étude de la pupille des Aliénés.) J. CHARDINAL and GASTÃO DE O. GUIMARÃES, *Arch. Brasil. d. Psych. Neur. e Med. Legal.*, Anno iv., Nos. 1 and 2, 1908.

IN this paper the authors state the results of an investigation into the pupillary phenomena of 1651 inmates of the Hospital for the Insane at Rio de Janeiro. The cases comprised 72 general paralytics; 364 cases of "alcoholism"; 116 of precocious dementia; 150 of epilepsy; 267 of hysteria; 7 of cerebral syphilis; 72 of paranoia; 12 of cerebral sclerosis; 170 of senile dementia; 45 of manic-depressive insanity; 17 of mania; 64 of lypemania; 51 of episodic delirium in degenerates; 37 of mental confusion; 63 of imbecility; 53 of feeble-mindedness, and 16 of idiocy. A noteworthy feature of the author's results was the frequency of abolition of light-reflex in almost every class of the insane. That the general paralytics gave the high proportion of 54.9

per cent. is perhaps not surprising, but the remaining categories naturally excite remark. Thus in the alcoholic class the proportion of male patients with abolition of the light-reflex was 18·2 per cent., dementia præcox 18 per cent., epilepsy 14·6 per cent., hysteria 18·1 per cent., senile dementia 28·5 per cent., manic-depressive insanity 19 per cent., mania and lypemania each 14 per cent., and episodic delirium in degenerates 18·3 per cent. On the other hand, anisocoria, or inequality of the pupils, which is generally believed to be more common in alcoholics, was only found in 6·5 per cent. of the authors' cases. The authors give useful references to the results of other investigators.

R. CUNYNGHAM BROWN.

TWO CASES OF DEMENTIA PRÆCOX. (Osservazioni anatomo-cliniche e cliniche intorno a due casi classificabili fra le demenze precoci.) BENIGNI and ZILLOCCI, *Riv. Sper. di Freniatr.*, Vol. xxxiv., Fasc. 1-2, pp. 23-56.

Two typical cases of dementia præcox are described in great detail, both clinically and pathologically. In both cases marked necrobiotic changes were found in the cortex, particularly in that of the frontal lobes and cerebellum, with degenerative vessel changes. The authors uphold the toxin hypothesis, and consider the more probable origin of the toxin to be the internal glands. They describe marked fatty degeneration of the parenchyma of the thyroid, suprarenal, and pituitary glands.

ERNEST JONES.

"DEMENTIA PRÆCOX IN INDIA." G. F. W. EWENS, *Indian Med. Gaz.*, June 1908.

MAJOR EWENS in this paper points out that there is in India a mental disorder of the period of adolescence which conforms in almost every particular with the "Dementia Præcox" of Kraepelin. This, he states, among Indian insanes is never recovered from: its duration is prolonged. It commences with mild excitement or depression, always accompanied by hallucinations. Its main features are "a peculiar tendency to grimacing, silly tricks of behaviour, a characteristic speech and manner, a peculiar combination of apathy, emotional dulness, and defect of volition, the whole passing inevitably into a characteristic weakness of intellect, in which very early defect of voluntary control over the sphincters and general feebleness of judgment and reasoning power contrast markedly with perfect retention of memory to a very late period." Ewens has no doubt but that such a disease exists in India as a clinical entity, and those who have had experience of insanity in that country will be found to agree with him. A detailed

description is then given of the general clinical features, modes of onset, the symptomatology and progress of the ailment, together with a note of the pronounced physical defects commonly met with among these persons, such as hypersalivation, polyuria, a tendency to diarrhoea, slow shallow respiration, etc. No treatment is of any avail, and the pathology is obscure. The complete notes of a typical instance of the hebephrenic variety is given, and this, it is interesting to note, is in a European born and bred in India. The hebephrenic type is the commonest, but Ewens records that he has two examples of the paranoid form in his asylum at present. The katatonic variety is also fairly common, and a description of it is promised in a future paper, which will be looked for with interest.

C. J. ROBERTSON MILNE.

THE FINAL STAGES OF DEMENTIA PRÆCOX. (*Endzustände (497) der Dementia Praecox. Gruppierung und Prognose der Dem. Praecox, I.*) JAHRMÄRKER, *Zentralbl. f. Nervenheilk. u. Psychiat.*, Ht. 13, S. 489.

THIS paper, read before the annual meeting of the German Society for Psychiatry, deals principally with the question as to how far the dementia præcox group can legitimately be split up. On this point the author is very sceptical as to the adequacy of our present knowledge. After some remarks on the importance of premonitory symptoms which, he insists, belong to the disease itself, he considers the various factors that account for the differences in clinical type observable. These he divides into:— (1) Factors which are accidental in that they lie outside the disease-process itself; such are personality, social and physiological differences, external factors, as alcohol, infection, etc.; the results of these may be temporary or permanent. (2) Factors which are part of the disease-process itself; rapid or chronic course, continuous or intermittent progress, etc.

The author accepts the new term, schizophrenia, that Bleuler has proposed, as a more accurate one for dementia præcox.

ERNEST JONES.

THE EYE SYNDROME OF DEMENTIA PRÆCOX. H. H. TYSON (498) and L. PIERCE CLARK (New York), *Journ. Amer. Med. Assoc.*, May 2, 1908.

A SYSTEMATIC attempt is made by the authors to analyse the significance of eye changes in 115 consecutive cases of dementia præcox. The work of Siglas in 1899, Meyer, Morro, 1900; Blin, 1905; and Dide and Ascot, 1901, is quoted and criticised as not having been sufficiently exhaustive to produce definite results.

The evidence that dementia præcox is of an autotoxic nature has been grouped as follows:—

1. "There is a coincidence of certain ocular symptoms with gastro-intestinal auto-intoxication, similar in many respects to these seen in typhoid, lead colic, and simple intestinal putrefaction."
2. "The urine in dementia præcox shows very defective elimination."
3. "Fully one-half of the subjects of dementia præcox die of tuberculosis."
4. "The co-existence of certain toxic dermatoses, such as certain types of erythema, vasomotor paresis, with chronic gastro-intestinal intoxication, is noteworthy."
5. "Some additional facts are at hand, as shown in the co-existence of psychic excesses, neurasthenia, etc., with states of auto-intoxication."
6. "The study of the blood in dementia præcox shows evidence of a toxic state."

The authors summarise their findings as follows:—

1. "They indicate that dementia præcox is attended by such an early and constant syndrome of alteration of disc, visual field, pupil and corneal sensibility, as to materially aid in diagnosing this psychosis. Consideration of the syndrome will particularly aid in the differential diagnosis of dementia præcox from the manic-depressive group, acquired neurasthenia, hysteria, and the various forms of imbecility and constitutional inferiority."
2. "The syndrome is a distinct contribution to the theory that dementia præcox is an autotoxic disease, and that the poison is primarily vascular, which finally induces neuronc degeneration. It points to a toxin of some sort, which is either a metabolic defect in the tissues (ductless gland defect), or, what seems more probable, that the poison is generated in the liver or in the gastro-intestinal tract itself."
3. "The syndrome is of prognostic value, as the severer grades of eye changes are found in the more rapidly deteriorating cases."
4. "Finally the optic nerve lesion is quite in accord with our best knowledge of the pathologic anatomy of dementia præcox, in other tracts of the brain (than the optic nerve, which itself may be counted an analogue). The early vascular changes in the brain ought to receive more serious investigation."

While the paper is ingenious and somewhat novel, actual experience seems to show that there are so many well-defined cases

of dementia præcox in which no ocular phenomena are found, and again, so many "congested discs, pallid discs, dilated veins, contracted arteries, low-grade perineuritis," etc., found in patients who are certainly not suffering from dementia præcox, that the deductions drawn are more likely to confuse than to clear the situation.

C. H. HOLMES.

**THE PSYCHO-ANALYTIC METHOD AND THE "ABWEHR-
(499) NEUROPSYCHOSEN" OF FREUD.** (*La méthode psycho-analytique et les "Abwehr-Neuropsychosen" de Freud.*) SCHMIERGELD and PROVOTELLE, *Journ. de Neurol.*, 5 and 20 avril, pp. 221 and 241.

THIS is one of the first attempts to introduce the knowledge of Freud's work into France, and consists in a short *exposé* of some of the principles underlying it, together with an abbreviated account of the analysis published by Freud in 1905. They give a few notes of four cases they have analysed, with confessedly very imperfect technique, and conclude by expressing their agreement with most of Freud's theory, though they do not follow him in many of his interpretations.

ERNEST JONES.

**NEW FACTS IN REFERENCE TO THE SERUM REACTION OF
(500) SYPHILIS IN GENERAL PARALYSIS BY WASSERMANN'S METHOD.** (*Les nouvelles données relatives à la séro-réaction de la syphilis dans la Paralyse Générale par la méthode de Wassermann.*) SOUTZO FILS, *Ann. Méd. Psychol.*, July-August 1908.

THE paper is a résumé of articles written on Wassermann's serum reaction in syphilis. Within the last two years much work has been done on this subject, and this goes to show that in the fluids of syphilitic, tabetic, and general paralytic patients there are specific antibodies. These antibodies are produced by specific antigens.

Bordet and Gengou, as well as Moreschi, Neisser, and Sach are credited with the discovery of the phenomenon known as the "fixation of the complement." This means that the antigen, meeting with the corresponding antibody, prevents hæmolysis. Wassermann and Bruck have demonstrated that not only bacteria, but also diseased extracts from organs, can be used as antigens to prove the presence of antibodies. It is this discovery which has been the basis of their experiments in syphilis, tabes, and general paralysis. It has been shown that the blood serum of monkeys treated with syphilitic material of monkeys or man, and mixed with an extract of syphilitic organs, gives rise to fixation of the complement. To obtain the reaction, Wassermann mixed an

extract of an organ of a syphilitic foetus with blood serum of a syphilitic patient. This serum had been rendered inert by being heated to 56° C. A complement, *e.g.* the fresh serum of a guinea-pig, was added, and finally red corpuscles of a sheep. It was found that in every case hæmolysis did not occur because fixation of the complement had taken place. This result was not obtained when the serum was mixed with an extract of normal liver, or when normal serum was used with a syphilitic liver. Levaditi and Marie have found that the reaction is obtained when one makes use of a normal liver extract, and other investigators were successful with lecithin, cholesterin, vaseline, bile salts, etc. Weil and Braun are opposed to Wassermann's teaching. They believe that auto-antibodies are produced by long-continued degenerative changes in the tissues.

Marie and Levaditi, in France, have made an extensive study of the reaction with cerebro-spinal fluid. They obtained a positive result in 73 per cent. of general paralytics. This is a smaller percentage than that of most German investigators, which varies from 80 to 88 per cent. They explain the relatively low proportion of positive results by the fact that their experiments have been carried out in early as well as in far advanced cases of general paralysis, and that in the former class the reaction is not so constant, because the antibodies are in smaller amount.

Levaditi and Jamanouki carried on the research with blood serum and cerebro-spinal fluid simultaneously in the same patients. They concluded that, with the latter, deviation of the complement is almost always present in general paralysis, very rarely present in cases with cerebral lesion, and never in other forms of insanity. Less constant results were obtained by them when blood serum was made use of.

Plaut, on the other hand, found that, where a reaction with the spinal fluid was obtained, a similar reaction was present with blood serum, but where the cerebro-spinal fluid gave a doubtful reaction, that obtained with the blood was always positive.

The writer concludes that deviation of the complement is evidence of acquired syphilis, but that the inability to obtain a positive result does not necessarily exclude syphilis. In any case it may be present at one time and absent at another.

A very complete bibliography is given.

R. DODS BROWN.

A MEDICAL STUDY OF CHATEAUBRIAND. (*Étude médicale* (501) *sur Chateaubriand.*) E. MASOIN, *Bull. de l'Acad. roy. de méd. de Belgique*, Tom. xxii., 1908, p. 24.

CHATEAUBRIAND derived his literary talent and "prodigious imagination" from his mother, and from his father his melancholy

and pride. A sister from an early age showed signs of melancholia and delusions of persecution, and probably committed suicide. The so-called confluent smallpox, from which he says that he suffered at the age of twenty-three, was, in Masoin's opinion, most probably urticaria, a highly probable hypothesis, from what is known of mistakes made during epidemic times at the present day, and from the fact that Chateaubriand, who was a remarkably handsome man, never presented any scars. Shortly afterwards he appears to have had an attack of melancholia which lasted four months, but the lamentations which he gave vent to throughout life were probably rather the outcome of a pose and of the force of habit than of any real conviction. So well-occupied a life would have been impossible had he suffered from the depression of persistent melancholia. During his stay in London, in 1793, he showed signs of tuberculosis, and was doomed by an eminent physician to an early grave. This gloomy forecast was not realised, since death, which was due to pneumonia, did not occur till fifty-five years later, at the age of eighty.

Frequent attacks of gout and rheumatism, as well as some liver trouble of indefinite nature, played only a secondary part in explaining his view of life. His acts and writings show that he possessed the erotic temperament in a high degree. The dark hints of his contemporaries as to his impotence lack confirmation. "This problem will remain an eternal riddle to excite the curiosity of investigators and the verve of physicians." In spite of his eccentricities and extravagances, his lively passions, his exuberant and sometimes ill-balanced imagination, Masoin thinks that there is nothing to justify Chateaubriand being ranked as a superior degenerate. "Physiology and psychology claim him as their own." The essay ends with a vigorous protest against the tendency originated by Lombroso and Nordau to stigmatise as degenerates some of the most illustrious men, too often on the strength of doubtful facts and apocryphal anecdotes.

J. D. ROLLESTON.

THE INSANITY OF MAUPASSANT. (*La folie de Maupassant.*)
(502) Z. LACASSAGNE, *Thèses de Toulouse*, 1906-1907, No. 717,
Rémond et Voivenel, Progrès Médical, May 30, 1908, p. 270.

THE writers reject the view that Maupassant was a victim of general paralysis, and think that he suffered from the chronic delirium described by Magnan and Sérieux.

Maupassant's heredity is of interest. His mother was a gifted woman, the friend of Flaubert, and of a highly nervous disposition. The father was a man of mediocre intelligence and of licentious tastes. Furthermore, Maupassant was predisposed to insanity by reason

of the sexual excesses of his early life, to which is to be added intoxication by ether, morphia, haschisch and cocaine.

The disease began in 1878, when he was 25 years old, and ran through the four periods of chronic delirium, uneasiness delusions of persecution, delusions of greatness, and, lastly, dementia. Death did not occur till 1893. Maupassant continued to write until the final period.

J. D. ROLLESTON.

THE MENTALLY DEFECTIVE IN PRISON. J. MILSON RHODES, (503) *Brit. Med. Journ.*, June 27, 1908.

THE writer in his article on the mentally defective under sentence has opened up a very important question for discussion. As a visiting justice he has been studying that *bête noir* of the alienist and the penologist, viz., the redevivist, the so-called habitual criminal, that the recidivist deserves far more attention than he has yet received appears to be proved by the fact that out of 182,645 prisoners convicted in 1906, 107,408 had previous convictions against them. Of this number

7,458			had been convicted four times.
5,612	"	"	five times.
17,093	"	"	six to ten times.
12,592	"	"	eleven to twenty.
10,700	"	"	above twenty times.

The "above twenty times" includes cases that have been one, two, and even two hundred times before the court. Some of these frequent offenders are specialists; one man will always steal fowls, another boots, another barrows, and another ladders. The two last should have directed attention long ago to the mental condition of the offenders, as ladders and barrows, being usually branded, they are very easy of identification, and therefore not likely to be stolen by any one who was *compos mentis*. The very reverse appears to have been the case. In a well-known work on penology we find the doctrine set forth that "such offenders must rather be dealt with by means of gradually but certainly cumulative sentences, and by subsequent police supervision." As far as I know neither of these will reform a feeble-minded prisoner. Both the so-called remedies have been tried for half a century, and both have been miserable failures. It is not the cumulative sentence, it is the indeterminate sentence, that is required to deal with this class.

The fact that out of 183,000 prisoners only 9200 were able to read and write well proves that the mass of prisoners are deficient in intellectual power, or that their environment—mental, moral, and physical—was so bad that their education has been confined to two branches—vice and crime.

Evil environment in youth is the commonest cause of men going wrong; the vast majority of prisoners have no idea of anything approaching intellectual enjoyment; their sole delight is in the lowest sensual pleasures—drink, gambling, and sexual vices. For a very large number of prisoners the short sentence is of no use; what we want and must have is a better classification of prisoners, which we are glad to see the Royal Commission on the Feeble-Minded are in favour of. We must have something more after the lines of the State Farm of the United States or the Dépôt de Mendicité of France and Belgium, colonies where they may have more liberty without more licence to do mischief or to work untold injury to the generation following; and to attain this end we must have power to detain the feeble-minded for an indeterminate period in colonies where they may be made more useful than in prisons.

That the number of feeble-minded prisoners is much larger than most people imagine is a fact, and it appears as if some of our prison medical officers fail to diagnose the true state of the case. Only a short time ago a young man who had been sent to a reformatory convicted of crime committed a murder two days after being released from gaol, and yet a prison medical officer said he was sane, against the opinion of two experts. Surely cases such as this prove the necessity for experts being called. The verdict of wilful murder against the man was quashed by the Court of Appeal, but that does not alter the fact that a man was sentenced to death for a crime the gravity of which he was mentally incapable of appreciating at the time it was committed.

AUTHOR'S ABSTRACT.

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Review of Neurology and Psychiatry

Original Article

A CASE OF INTRACRANIAL TUMOUR.

By EDWIN BRAMWELL, M.B., F.R.C.P.,
Assistant Physician to the Edinburgh Royal Infirmary.

THE case which is here recorded presents the following features of interest:—(a) The patient suffered for four and a half years from epileptic attacks; (b) optic neuritis of slight degree constituted the only objective sign of organic disease; (c) death occurred after five days' illness, the patient having been absolutely free from symptoms apart from fits for six months previously; (d) a series of general convulsive fits occurred five days before the fatal termination, and continuous movements of protrusion and retraction of the tongue were observed shortly before death; (e) a hæmorrhage into a large glioma situated in the right frontal lobe was the cause of death.

Report of Case.—C. M., a lady's-maid, aged 28, was seen by the writer at Leith Hospital on 24th November 1906. She stated that she had suffered from fits for three years, that she had had severe headaches for the last seven or eight weeks, that three weeks ago she vomited for two days on end. She could not recollect ever having vomited before, nor had she done so since. From her medical attendant it was ascertained that Dr J. V. Paterson, who had examined the optic discs a few days previously, had noted the presence of double optic neuritis.

Upon inquiry it was ascertained that she had suffered from headaches, which appeared to be of the megrim type, since childhood. Her health otherwise had been good, and, with the exception of an attack of typhoid fever when nine years of age, she had had no illnesses of importance. There was nothing in the history suggestive of syphilis, nor was any evidence of that disease detected upon examination. The family history was unimportant, saving for the fact that her father had died, aged fifty-six, from a tumour of the leg which recurred after operation. No history of tubercle among the relatives was obtained.

The first fit occurred in February three years ago, between seven and eight o'clock upon a Saturday evening. She was in service in London at the time, and had been feeling particularly well. She remained unconscious until the following Tuesday, having nineteen fits in all during that time. Four months later she had another fit, and again another ten months after this. She stated that she had had about fifteen fits from that date up to November 1906. She had taken bromides regularly, but without any apparent benefit. In June she went to America, where she had a fit, another occurring about the beginning of October. No accurate account of the character of the fits could be obtained. She stated that they came on at any time of the day, that she had no warning, that she used to fall down unconscious, that she believed she was convulsed, that she had never hurt herself in falling, that she passed water sometimes when unconscious, but had never bitten her tongue. When in America, towards the end of September, she suffered from headache, which was distributed all over the head, and extended down the back of the neck. So severe was the headache that for several nights she obtained no sleep. The doctor who saw her at the time attributed the headache to sunstroke. Lately the headache has gradually improved; occasionally, however, she has a great deal of pain over the eyes, while she states that at times she momentarily loses her sight.

The patient is a well-built, though somewhat poorly-nourished girl. Height, 5 ft. 6 ins.; weight, 8 st. 7 lbs. She is not anæmic. She is intelligent, and presents no obvious mental peculiarity. Her memory, she states, is fairly good, and this is borne out upon examination. She speaks fluently, and never appears to be at a loss for a word. She understands everything

that is said to her, and has no difficulty in reading or expressing herself in written language. She is right-handed. There is no history of left-handedness among her relations. Articulation is perfectly distinct and phonation unimpaired. Although she states that at times a cloud seems to pass over her eyes, upon examination vision is found to equal $\frac{6}{7}$ with either eye. There is a slight degree of optic neuritis, the appearances being approximately the same in both eyes. No alteration in the fields of vision is detected upon rough examination. She has had no diplopia. She hears a watch at about two feet from either ear. There have been no subjective auditory phenomena. There has been no discharge from the ears. She recognises the usual test specimens for smell with both the right and left nostril, although she cannot name them. There is no note as to the sense of taste.

There is no ptosis, the optic axes are parallel, the ocular movements are perfectly satisfactory, and there is no nystagmus. There is no weakness of the jaw muscles, and no sensory disturbance in the distribution of the fifth nerve. There is no facial asymmetry, and the facial movements are satisfactory. No difficulty in swallowing. The palate moves well. The tongue is protruded in the middle line, and shows no wasting.

The movements of the limbs are all well carried out. Dynamometer: Rt. 70, Lt. 55. Co-ordination is perfect, and there is no tremor and no astereognosis. There is no defect in the gait.

The knee jerks and ankle jerks are brisk and equal. No ankle clonus. The abdominal reflexes active and equal. The plantar reflexes are of the flexor type.

The skull is symmetrical. There is no local prominence and no area which is tender on pressure. Nothing abnormal is detected on auscultation and percussion of the cranium.

The heart is of normal size and the sounds pure. Nothing abnormal is found upon examination of the lungs and abdomen. No *striæ gravidarum*. The urine contains neither sugar nor albumen.

On 28th November the patient was again examined by the writer. It is noted that she had had, since last seen, some headache on the top of the head, which, however, had not been severe. It is also stated that there is no tremor of the hands.

14th February 1907.—The patient remained fairly well until 4th January, when she took another situation as lady's-maid. After she had been in service for a week there was a return of the headache, which was severe and situated over the top and back of the head, and was accompanied by vomiting which lasted throughout a whole night. Last week she had another bout of vomiting which persisted all day. Headache has been present every day, though not severe; it often feels merely "like a stiffness" at the back of the neck, she says. Upon examination there is nothing to add to the previous notes excepting the presence of (?) slight right facial weakness. There is no tremor of the hands.

Patient was admitted for observation to the Royal Infirmary under the care of Dr Byrom Bramwell on 15th February, and remained in the ward until 25th March. During the whole of this time she was free from headache and had neither giddiness nor vomiting. Her eyes were repeatedly examined during her stay in hospital. The acuity of vision remained at $\frac{5}{6}$. The fields of vision were slightly contracted. The optic neuritis did not progress. The temperature remained sub-normal throughout. The pulse ran from 68 to 96. It was noted that she was rather irritable at times and that her memory was slightly defective. She had no fits during her stay in hospital. The slight degree of weakness of the right side of the face is again referred to. The patient was given 30 grains of potassium iodide three times a day over a considerable period. The question of operation was carefully considered, but decided against, since the headache had disappeared and the optic neuritis had not increased, while no new symptoms had developed.

The patient died on 18th November 1907. Her medical attendant kindly sent me the following account of her illness during the five days which preceded death.

"During the last six months, ever since she left the Infirmary, she has had no headaches, and her eyesight seemed to improve. On the day she took ill (13th November) she said to a friend that she had never felt better in her life. She was a great sleeper, went to bed at 10 P.M. and did not rise till 11 A.M., sleeping the whole time, except when wakened to take her breakfast. She was always very bright, except on days following the fits.

"At 11 P.M., 13th November, had a fit during her sleep (has

not had fits since July, when she had two in one week). Two other fits occurred before 12.30 A.M., when a sixth of a grain of morphia was given hypodermically. Three more fits during the next hour, but no more till 2 P.M., 14th November. During the forenoon she was sick, and heavy retching at times, but had risen from bed in order to go to bathroom. At 2 P.M. fits recurred, and she lapsed into unconsciousness, having fits almost every quarter of an hour. They began by her opening her eyes and staring, then a general convulsion, with head retraction and turning of face over left shoulder, followed by clonic movements of whole body. Stertorous breathing and bubbling râles in bronchial tubes were continuous. Heart's action vigorous and pulse regular."

On Friday, 15th November, the patient was seen by the writer at 11 A.M. She was comatose. Babinski's sign was present on the left, not on the right side. The pupils were equal, and of medium size. There was no deviation of the eyes or head. There had been no fits for some hours previously.

"The fits recurred at 3 P.M., twelve taking place in a few hours. No special order of movements, but the left arm and leg were not moved to the same extent as the right. Fits ceased later in the evening, and did not return. On Saturday evening there was noticed continuous movement of the tongue and lower jaw. The tongue was protruded, then retracted, and swallowing movements took place, but fluid placed in the mouth ran out at the sides. Temperature, 105; pulse, 110."

17th November.—"Has lain quietly all day. When spoken to, opens her eyes, but without sign of recognition. Has not spoken since 14th November at 2 P.M. The last fit occurred on 15th November, in the evening."

18th November.—"Died at 8 A.M."

Post-mortem.—Permission was obtained to examine the head. Nothing abnormal was noted until the dura was opened and the brain removed. It was then observed that the second and third frontal convolutions on the right side were much wider than those on the left, and that the brain in this region was somewhat fuller, and seemed slightly more resistant on palpation.

After the brain had been hardened in 10 per cent. formalin, a series of transverse vertical sections were made and examined.

In a transverse vertical section passing through the genu of

the corpus callosum (corresponding to No. 37 of Dejerine's series) the right frontal lobe is seen to be distinctly larger than its fellow. The lower end of the superior longitudinal fissure is pushed up and to the left. The greater part of the centrum ovale on the right side is evidently occupied by a tumour which is not encapsuled, and which, both in its colour and consistence, closely resembles the normal white matter of the brain. The grey matter of the second and third frontal convolutions is quite indistinguishable from the subjacent white matter. On the other hand, the line of demarcation between cortex and sub-cortex is clearly defined in the case of the first frontal and the upper temporo-sphenoidal convolution in this section. A large recent hæmorrhage, more or less circular in outline, and measuring from 2 to $2\frac{1}{4}$ inches in diameter, is situated in the right centrum ovale. Although the hæmorrhage has found its way to the right lateral ventricle, no blood was found in the other ventricles of the brain. In a transverse vertical section immediately posterior to the tip of the temporo-sphenoidal lobes (corresponding to No. 55 of Dejerine's series) the cortex of the first, second, and ascending frontal convolutions is seen to be clearly distinguishable from the subjacent white matter. The nucleus caudatus and internal capsule have been destroyed, but the lenticular nucleus has not been implicated in the hæmorrhage. Upon microscopical examination the tumour was found to be a very vascular glioma, the blood vessels having very thin walls.

Remarks.—The possibility of gross intracranial disease was first suspected by the patient's medical man in November 1906, when he obtained the history of the severe headaches from which she had suffered two months previously when in America. The circumstance that she had been troubled with headaches ever since childhood probably led the patient to attach less importance to this symptom than she would otherwise have done. The detection of optic neuritis by Dr Paterson suggested the probability of organic disease.

When I examined the patient on 24th November I made the diagnosis of "? Intracranial Tumour," but detected no signs which enabled me to locate the growth, should one exist. No description of the fits was obtained; the headache, which had not been unilateral, had been chiefly vertical and down the back of the neck. The dearth of positive signs and symptoms sug-

gested to me the possibility of a frontal growth. There was, however, no defect of speech and no obvious mental impairment. I noted that vision was $\frac{5}{8}$ in both eyes; that there was no tremor of the hands; that smell was normal; and that the abdominal reflexes were active and equal. After examining the patient on 13th February, I wrote to her doctor: "There must, I think, be an intracranial tumour, but I do not feel at all sure as to its exact location. I thought to-day there was just a suspicion of a little weakness on the right side of the face. I agree with you that the best thing to do would be to have her under observation in hospital, when she will be put on a thorough course of iodides, while her acuity of vision is at the same time carefully watched." On 4th March I wrote: "She has been almost free from headaches since admission, and we have obtained no further data bearing on the position of the growth. The acuity of vision is perfect, and there is only one dioptré of swelling of the discs. We shall test the visual acuity again carefully, and if there is no deterioration since admission, I think there is nothing to be gained by keeping her longer in hospital in the meantime."

The patient was discharged on 25th March, and I heard nothing of her until her doctor communicated with me on the morning of 15th November, asking me to see her with him.

Although general epileptic attacks are far from uncommon in the later stages of cases of intracranial tumour, the occurrence of a series of fits, practically amounting to a condition of status epilepticus as the initial symptom, is very unusual. The most reasonable explanation of the initial fits and coma in this case would seem to be, in the opinion of the writer, a hæmorrhage into the gliomatous tumour, which up to that date had remained quiescent in the sense that it had given rise to no symptoms. The recent hæmorrhage into the tumour, which was found post-mortem, and which was undoubtedly the cause of death, lends support to this view. The writer has met with three other cases in which a hæmorrhage into an unsuspected tumour was attended with fatal results.

A case observed by Bruns and Oppenheim, and referred to by the latter writer,¹ is of interest in connection with the case above described. "A woman, aged 39, had had epileptic

¹ Oppenheim, *Geschwülste des Gehirns*, Zweite Auflage, Wien, 1902, S. 68.

attacks now and again from the time she was 17 years of age. In 1886, after the birth of her last child, for several weeks these attacks were more frequent. Latterly the attacks became less frequent, their place being taken by very severe attacks of megrim. In April 1892 a definite diagnosis of brain tumour was made. Post-mortem a large sarcoma was found in the right frontal region."

Jastrowitz, quoted by the same writer, describes the case of a patient, an inmate of an asylum, who for a number of years had suffered from epilepsy, and who was free from attacks during the last few years of his life. At the autopsy a tuberculous tumour was found at the apex of the right frontal lobe.

The movements of protrusion and retraction of the tongue are of interest, although the description of them is meagre, since they suggest irritation of the centre in the frontal lobe described by Horsley and Beever,¹ stimulation of which is followed by similar movements.

It is interesting to note that at no time while this case was under observation were the homolateral tremor and the loss of the abdominal reflex on the side opposite to the growth, signs shown by Grainger Stewart to frequently accompany a frontal tumour,² observed though repeatedly looked for.

Abstracts

ANATOMY.

THE MIDDLE CELLS OF THE GREY MATTER OF THE SPINAL
(504) **CORD.** J. H. HARVEY PIRIE, *Proc. Roy. Soc. Edin.*, Vol. xxviii., Pt. 8, No. 36.

THE middle cells are present throughout the whole length of the spinal cord. They are situated in the middle portion of grey matter at the junction of anterior and posterior cornua; but they sometimes also extend into the regions usually occupied by the motor cells, by the intermedio-lateral tract and by Clarke's column. They cannot be sharply separated from the "scattered cells" at the base of the anterior horn, nor yet from the small cells at the base of the posterior horn. They have a more or less typical

¹ *Phil. Trans. Roy. Soc.*, 1894, Vol. 185, B.

² *Rev. of Neurol. and Psychiat.*, 1906, p. 809.

arrangement in each segment. This is described in detail and figured in the paper. In the cervical enlargement they appear as a broad band of cells extending from the formatio reticularis to about the anterior grey commissure. In the dorsal region they are fewer in number, and may be found scattered irregularly, or more often occurring in small groups either centrally, about on a level with the central canal; or post-central, between Clarke's column and the reticular group of the intermedio-lateral tract. In the lumbo-sacral enlargement they are very abundant, and their field extends until in the lower sacral segments they occupy in addition practically the whole anterior cornua. At all levels, as studied in serial sections, their distribution is seemingly erratic, there is no segmentation (as Argutinski described) like that so well seen in the intermedio-lateral tract. Sometimes they are dotted about singly, or they may be found as little cell groups, but never very closely packed. They may be divided apparently into three groups:—(1) The middle cells proper or central cells, occurring chiefly in the central area, and very similar individually to cells of the apical group of the intermedio-lateral tract; (2) anterior or scattered cells in the base of the anterior horn, fewer in number and seldom grouped, but larger individually; (3) post-central cells, lying chiefly between Clarke's column and the reticular group of the intermedio-lateral tract, mostly smaller in size than the others.

The course and termination of the cell processes have not been ascertained.

AUTHOR'S ABSTRACT.

PHYSIOLOGY.

THE PHYSIOLOGY OF THE PONS AND CORPORA QUADRI-
(505) **GEMINA.** (Beitrag zur Physiologie der Varolsbrücke (Pons Varolii) und der Vierhügel (Corpora bigemina).) OSWALD POLIMANTI, *Arch. f. Anat. u. Physiol.*, Heft iii. and iv., 1908, p. 271.

THE author records experiments on dogs and cats where solutions of morphine, curare, quinine, or cocaine were injected in the region of the pons and corpora quadrigemina. The solutions were tinged with thionin, whereby the parts affected could be ascertained on *post-mortem* examination.

Morphine and curare, so injected, are found to act as convulsants; quinine similarly, but in a less degree. The convulsions produced are not bulbar in their origin, since the bulbar respiratory centre is not paralysed as it is in cases of bulbar poisoning through strychnine or nicotine. The convulsive attacks have a relation to

acoustic and visual stimuli, as is to be expected from the anatomical connections of the part of the brain affected. They are of a tonic-clonic character. Morphine and curare give rise to genuine epileptic attacks, and we have to do here with epilepsy of sub-cortical origin, quite independent of the cortex. The stimulation of pons and corpora quadrigemina is so great that the inhibitive influence of the cortex is entirely cut out. The animals are often observed to run in a circle, and rotatory movements towards the side opposite to the injury are seen.

Emotional phenomena are induced. The animals show great excitement, springing and barking, and making aggressive movements, as though they saw spectres.

The author brings his results into relation with those of Sherrington, where a dog whose brain is separated from the spinal cord exhibits the customary movements expressive of fear, pleasure, anger, and disgust; and discusses Bechterew's findings with regard to the independence of emotion and emotion mimicry. He concludes, from his own experiments and those of others, that all the grey masses at the base of the brain take part in the various expressions of primitive emotions, and recalls Goltz's experiment where psychical manifestations were given by a dog whose brain, including almost the whole of the basal ganglia and part of the mid-brain, was destroyed.

Many animals, after injection in the pons or corpora quadrigemina, exhibit raising of the hair, especially of the back and tail. It is probable that we have here a centre presiding over the *arrectores pilorum*, and its action appears to be independent of the emotional condition of the animals.

In all the operated animals the reflex activity is heightened. A knock on the box in which the animal is kept suffices to call forth vigorous motor reactions. It follows that an inhibitory centre must be present in the pons or corpora quadrigemina, or that damage to these parts prevents the higher inhibitory centres from exercising their function.

Rhythmical contractions of the face muscles, observed in the animals, are referred to damage of the 7th nerve, and nystagmus and other abnormal movements of the eyes to involvement of the 6th and 8th nerves.

A great flow of saliva follows on the injection of morphine, curare, or quinine, which is independent of the epileptic attacks, being due to stimulation of the chorda tympani. A transitory flow is caused by cocaine.

Many animals after injury to the pons or corpora quadrigemina exhibit alteration of the voice. This supports the view that a centre for voice production is present in this region. In opposition to Pussep, erection of the penis has not been observed.

The experiments show that there exist two motor centres, one situated in the mid-brain, in relation with visual and auditory impressions, and the other in the pons, in relation with acoustic-facial reception, with perception of equilibrium, etc. After injury to the pons, pendulum-like movements of the head and a characteristic position of the neck are seen, very similar to appearances presented after lesions of the semi-circular canals or cerebellum.

The movements caused by stimulation of those parts of the brain are produced by direct excitation of motor elements which have their origin there.

After injection of cocaine in the pons or corpora quadrigemina, movements are carried out after a certain period, but co-ordination and regularity are wanting until the action of the drug passes off—that is to say, until these centres resume their normal function. The cortical centres are not able to produce harmonised movements without the aid of these lower motor centres.

W. A. JOLLY.

ON THE FUNCTIONS OF THE NEUROGLIA. LUGARO, *Arch.* (506) *Ital. de Biol.*, T. 48, f. 3, 1908, p. 357.

THIS paper is based in part on the work of Cajal. The conclusions Lugaro arrives at may be stated as follows:—

1. The *fibres* of the neuroglia support and add elasticity to the nerve tissue.

2. The *protoplasm* of the neuroglial cells acts as an insulating medium, confining nerve stimuli to their proper line of neurones and preventing leakage of energy.

3. The protoplasm of the neuroglial cells acts as a filter for toxic products of normal metabolism and renders them innocuous. The toxic products may be in part in the blood and in part in the nerve elements, and possibly the neuroglial cells prevent toxic agents in the blood from damaging the neurones. It is probable that nervous excitations at articulations of neighbouring neurones may be of a chemical nature, and it is possible that here also the neuroglial cells may take some part in the process, perhaps by eliminating waste products which may be formed.

4. In pathological conditions the neuroglial cells acquire perverted activity, and may become the source of toxic substances. The importance of this in many affections of the nervous system is obvious, if somewhat theoretical.

5. The neuroglia may exercise nutritive or chemical influences in the process of development, although the part played is difficult to define.

ROBERT A. FLEMING.

THE CENTRE FOR THE SUBMAXILLARY GLAND. (Vom (507) *Centrum der Submaxillar Drüse.*) JULIAN SOLOMOWICZ, *Neurol. Centralbl.*, Aug. 1, 1908, S. 724.

THE researches of Grützner and Schlapowski and Hermann have demonstrated that the reflex centre for the secretion of saliva exists in the medulla.

Beck, experimenting on dogs, using the method of cross section of the medulla, came to the conclusion that the reflex centre for the secretion of saliva remained intact when the facial nucleus was uninjured, while when it was damaged the secretion was arrested.

Kohnstamm divided the chorda tympani, central to the submaxillary ganglion, in two dogs and examined the medulla. He found the nuclei of nerves 5, 7 and 10 intact, but, on the contrary, numerous degenerated cells over a wide area, especially on the side opposite to that of operation, at the level of the facial nucleus.

The author has examined histologically the medulla of two dogs in which twenty-one and twenty-six days previously the submaxillary gland had been excised. He arrives at the following conclusions:—(a) The centre for the submaxillary gland is formed of cells which are distributed in the region of Deiter's nucleus; only very few are found in the substantia reticularis; (b) the cells which form the above-mentioned centre are found in both halves of the medulla, with slight predominance on the side of the corresponding gland.

EDWIN BRAMWELL.

ON THE QUESTION OF INHIBITORY FIBRES IN PERIPHERAL NERVES. (508) *NERVES.* (Zur Frage der hemmenden Fasern in den Muskel-nerven.) F. W. FRÖHLICH, *Arch. f. Anat. u. Physiol.*, Heft iii. and iv., p. 392, 1908.

THE author discusses the question as to whether inhibitory processes are conducted from the centre to the periphery, or whether inhibitory fibres are present in the peripheral nerves, and concludes that there is no proof of the presence of such fibres in peripheral nerves.

Nikolaïdes and Dontas have published experiments which lead them to the belief that inhibitory fibres to the gastrocnemius muscle are contained in the ninth spinal nerve of the frog. Their results have been subjected to criticism by Woolley and by Fröhlich, who point out that the former authors have overlooked the spread of current from stimulated to non-stimulated roots, and that the inhibition obtained by them is a special case of the phenomena described by Wedensky and others.

The author compares the curves of Nikolaïdes and Dontas with those given by Hofmann and Amaya.

W. A. JOLLY.

**NOTE ON THE SUPPOSED EXISTENCE OF VASO-CONSTRICTOR
(509) FIBRES IN THE CHORDA TYMPANI NERVE.** W. M.
BAYLISS, *Journ. of Physiol.*, Vol. xxxvii., 1908, p. 256.

THE chorda tympani is usually regarded as a typical vaso-dilator nerve. Fröhlich and Loewi state that, after the administration of amyl nitrite or sodium nitrite, stimulation of the chorda is attended with *diminution* of the blood-flow from the vein of the submaxillary gland. They conclude from this that there are present in the chorda vaso-constrictor fibres whose effects become more manifest after the injection of nitrite. These results are not confirmed by Bayliss, who fails to find evidence of such constriction after the use of nitrite, or in the general excitation of asphyxia, where it might be expected, were vaso-constrictor fibres present.

With regard to the effects of pilocarpine, if arterial constriction is caused by this drug, it should be referred to the action of the drug upon the terminations of the sympathetic constrictors in the gland, or directly upon the muscular coat of the arterioles, rather than to central excitation of constrictors contained in the chorda.

The results obtained by Fröhlich and Loewi are explained by Bayliss as due to escape of the exciting current to surrounding tissues, producing contraction of muscular structures under the vein and thus causing its obstruction. Further, from experiments upon the cervical sympathetic in the cat, it appears that the method of investigation adopted by these observers—stimulation after injection of nitrite—is not likely to reveal the presence of vaso-constrictors in a nerve.

W. A. JOLLY.

**THE EXCITATION OF VASO-DILATOR NERVE FIBRES IN
(510) DEPRESSOR REFLEXES.** W. M. BAYLISS, *Journ. of Physiol.*,
Vol. xxxvii., 1908, p. 264.

ON stimulation of the central end of the vagus in the cat, there is acceleration of blood-flow through the submaxillary gland of the opposite side, on which the cervical sympathetic has been cut. There is thus excitation of the vaso-dilator fibres of the chorda tympani in a depressor reflex. When the chorda is cut and the cervical sympathetic left intact, there is also increased flow through the gland. This must be due to inhibition of constrictor tone.

At first sight it appears that we have to do here with undoubted reciprocal innervation in vaso-motor reflexes, but it may be objected that the inhibition of constrictor tone represents the

result of excitation of the true depressor fibres in the vagus, while the excitation of dilators may be due to a special reflex following on excitation of afferent fibres from the alimentary canal, and connected with the secretion of saliva.

Experiments are described which show that it is possible on stimulating the central end of the vagus in the cat to obtain excitation of dilators although no secretion is present. The excitation of the central end of the anterior crural nerve produces vaso-dilatation in the gland vessels, with a considerable flow of saliva. The increased flow does not of itself necessarily imply excitation of vaso-dilator nerves, since the effect might be due to the production of metabolites from the secretory cells. Vaso-dilatation is not obtained in the gland on stimulation of the vagus below the entrance of the fibres from the aorta.

Experiments were performed in which the depressor nerve was excited in the rabbit. The effect of changes in the blood pressure is diminished by the use of a mercury compensator connected with the abdominal aorta. This compensator consists of a Voit's flask and mercury reservoir, and by its action blood, rendered non-coagulable by hirudin, is automatically removed or returned as the blood-pressure rises or falls. On stimulation of the depressor nerve there is a marked acceleration of blood-flow through the sub-maxillary gland, whose vaso-constrictor nerves have been cut, due to excitation of the chorda dilator fibres.

The experiments show that the action of the depressor nerve brings about not only inhibition of tone of the vaso-constrictor centre, but also excitation of vaso-dilator nerves, and support the view that there exists in vascular reflexes a process akin to Sherrington's reciprocal innervation in voluntary muscle reflexes.

W. A. JOLLY.

OBSERVATIONS ON THE ACTION OF NITRITES AND ATROPIN

(511) **ON THE AUTONOMIC NERVOUS SYSTEM.** A. FRÖHLICH

and O. LOEWI, *Archiv für Exper. Pathol. und Pharmathol*, Bd. 59, H. 1, 1908, p. 34.

FRÖHLICH and Loewi have, in the Pharmacological Institute of Wien, conducted for some time a large series of observations with various medicinal substances, attempting to isolate the precise and particular portion of the nervous system on which they have a specific action.

This has been done previously for several other drugs—ergotin, adrenalin, and pilocarpin—and their observations were concerned with nitrites and atropin.

They first of all discuss the innervation of the various organs,

glands, etc., and distinguish broadly between autonomic and sympathetic fibres to these parts. The former has a cranial and a sacral portion, the former springing from the mid-brain and medulla, and the latter from the sacral portion of the spinal cord. The sympathetic fibres arise in sequence from the thoracic and lumbar cords. From these two systems the various organs have a four-fold innervation—two sets of fibres from each—autonomic motor and autonomic inhibitory, sympathetic motor and sympathetic inhibitory.

Nitrites.—Their observations were made chiefly on cats and dogs. The autonomic and sympathetic fibres to various organs—*e.g.* pupil of eye, tongue, penis, bladder, etc.—were dissected out. The action of the nerve was ascertained by section and electrical stimulation of the cut end. This was followed by the intravenous injection of nitrite of soda in varying doses, and the result watched.

They found that the nitrite has no effect on either group of sympathetic fibres, nor on the autonomic motor fibres. A marked and regular effect was obtained on the autonomic inhibiting fibres. After the injection of the nitrite the impulses were prevented passing and the action of the nerve fibres abolished. This they found without exception in all the nerves to the various organs on which they made observations.

Atropin.—Numerous and exact observations were made with atropin in continuation of previous work of their own and of others. The method of procedure was the same as with the nitrites. The nerve was separated, then cut, and the injection of atropin followed.

They came to the conclusion that atropin, like the nitrites, has no effect on the two sets of sympathetic fibres. No effect was observed on the autonomic inhibiting fibres. But following the injection of atropin the autonomic motor fibres are put out of action and no contraction obtained. In the case of atropin, however, two exceptions were observed. The autonomic motor nerves to the rectum and bladder continued to respond, and a contraction was obtained after the injection of atropin. Atropin has no paralytic action on the nerves to these organs. They consider that atropin has exclusively and regularly an action on these autonomic fibres.

Both atropin and the nitrites exercise an action only on the autonomic and not on the sympathetic fibres, the one on the motor and the other on the inhibitory fibres.

EDWIN MATTHEW.

PSYCHOLOGY.

AN EXPERIMENTAL STUDY OF SLEEP. BORIS SIDIS, *Journ.* (512) of *Abnorm. Psy.*, Aug.-Sept. 1908, p. 170.

THE third and final instalment of Dr Sidis's study of sleep deals with the nature of reactions in sleep, the relation of cell energy to sleep, and motor consciousness and sleep.

With respect to the nature of sleep, Dr Sidis agrees with Claparède's biological view that sleep is a function of defence, an instinct which has for object the prevention of exhaustion. Sleep is not a kind of narcosis of the system by the poison of fatigue products; it is a state into which we fall at will, and out of which we can awake at pleasure.

In common with most modern psychologists, the author lays great stress on motor consciousness as being at the very heart of personality. "We are what we can accomplish."

A useful bibliography is appended to the paper.

MARGARET DRUMMOND.

THE STATE OF THE BRAIN DURING HYPNOSIS. WILLIAM (513) M'DOUGALL, *Brain*, Vol. xxxi., 1908, p. 242.

THE aim of the paper is to render clearer and more definite the "theory of cerebral dissociation," and to show how explanations of many of the phenomena of hypnosis may be deduced from the theory in the more definite form proposed. It is contended that hypnosis is closely allied to the state of sleep, and that while the state of the brain is very similar in these two conditions, its state during hypnosis differs from that obtaining during sleep in the possession of certain positive characteristics. The first step must therefore be to obtain a satisfactory conception of the state of the brain during sleep; this may be arrived at by denying it those positive characters which constitute the waking state.

The principal difference between the sleeping and the waking brain is that the latter is suffused with free or active energy which, though constantly being used, is constantly renewed through the stimulations of the sense-organs and the activity of the great functional dispositions within the brain. The presence of this charge of varying potential during the waking state maintains the synapses of the brain, the principal seats of the resistances which delimit the neural systems of which it is composed, in a condition of lowered resistance, so that interplay between all parts is free

and easy. All factors that tend to diminish the quantity of free energy present in the brain (*e.g.* withdrawal of sensory stimuli) or directly to raise the resistances of the synapses (*e.g.* action on them of products of metabolism), therefore tend to render less easy the interplay between neural systems. Any considerable degree of such general increase of synaptic resistances constitutes a state of cerebral dissociation. Such a state obtains during sleep and hypnosis. Hypnosis differs from sleep in that one group of neural systems, all those concerned with the presentation of the operator, are kept in a state of excitation, while the rest of the brain falls into quiescence. The one active group then serves as a channel through which other systems may be brought into isolated activity; and each one then, functioning in the absence of the partial inhibitions normally exerted during the waking state by the dispositions of rival ideas, operates with abnormal intensity.

AUTHOR'S ABSTRACT.

PATHOLOGY.

SOME POINTS IN THE HISTOLOGY OF LYMPHOGENOUS AND (514) HÆMATOGENOUS TOXIC LESIONS OF THE SPINAL CORD. ORR and Rows, *Journ. of Ment. Sc.*, July 1908.

THE following constitute the principal points of difference between the above two types of lesion :—

When the spinal cord is infected by toxins *via* the lymph current ascending in the perineural sheath, the posterior columns show degeneration in the root entry zones. The fibres around the median septum remain normal. With increased toxicity of the lymph entering the cord there is a greater tendency to diffusion, so that changes are found round the cord-margin and along the septa. The morbid process spreads from below upwards. The Marchi method gives a positive reaction. In general blood toxæmia the cord lesions are very different. The degeneration is diffused. In the posterior columns it affects the fibres round the median septum first, leaving the root entry zone intact. The basis bundles, the crossed pyramidal tract and the fibres in the grey matter are attacked, as is also the cord margin to a slight extent. The lesion is most marked in the cervical and upper dorsal cord, and spreads downwards to the lumbar region.

Marchi's method gives a negative reaction. The morbid change consists in a slow atrophy of the myelin, but with increased toxicity of the blood this may be swollen and varicose.

The system lesions in tabes dorsalis and those occurring in the

cord and cranial nerves of general paralysis of the insane fall into the lymphogenous group; the cord lesions in acute insanity, leukæmia, pernicious anæmia, Addison's disease, metallic poisonings, etc., fall into the hæmatogenous group.

AUTHOR'S ABSTRACT.

CELLULAR LESIONS OF THE ANTERIOR HORNS IN NERVOUS ARTHROPATHIES. (515) **VOUS ARTHROPATHIES.** (*Les lésions cellulaires des cornes antérieures de la moëlle dans les arthropathies nerveuses.*)
ETIENNE and CHAMPY, *L'Encéphale*, May 1908, p. 369.

THE authors examined carefully, with modern methods, the anterior horn cells in a case of tabes, of syringomyelia, and of Aran-Duchenne muscular atrophy respectively, each one of which was associated with an arthropathy.

1. Arthropathy of the right shoulder in a tabetic. In this case throughout the cord the anterior horn cells were affected to a varying extent at different levels. Chromatolysis was found, its first stage being some diffusion of the Nissl bodies, becoming complete throughout the cell, with vacuolation, disappearance of the nucleus, and eventually neuronophagia. In the cervical cord not a single normal anterior horn cell was found. On the right side, corresponding to the arthropathy, specially in the postero-external groups, the cells were in an advanced state of degeneration; many had entirely disappeared.

2. Arthropathy of the left shoulder in a case of syringomyelia. In this case there was a glioma of the cervical cord, with cavity formation, and the anterior horn cells were much affected, all, more or less, being in a state of chromatolysis. The cellular lesions were bilateral, but much more intense on the left side than on the right, and again the chief area affected was the postero-external group.

3. In a rare case of arthropathy of the shoulders in progressive muscular atrophy, the authors found all the cells of the cervical cord affected, in particular those belonging to the postero-external groups: the latter showed intense chromatolysis, with complete "pulverisation" of the Nissl bodies, but without vacuolisation or neuronophagia.

Nervous arthropathies are caused by a disturbance of reflex trophic equilibrium, as a result of lesions of the anterior horn cells. These lesions may be determined by alteration of the sensory influx from disease of the centripetal protoneurons, or by disease of the sensorimotor branches of these protoneurons which pass to the anterior horns; or, again, the anterior horn cell lesion may be primary, as in progressive muscular atrophy, and may of

itself determine the arthropathy. It is conceivable, further, that these changes may be induced by alteration in the trophic influence coming to the cells from levels higher up.

S. A. K. WILSON.

CLINICAL NEUROLOGY.

THE RELATIONSHIP OF THE SO-CALLED FAMILY DISEASE (516) TO A PREMATURE PHYSIOLOGICAL SENESCENCE LOCALIZED TO CERTAIN ORGANIC SYSTEMS, CONSIDERED WITH SPECIAL REFERENCE TO THE NERVOUS SYSTEM. F. RAYMOND, *Lancet*, June 27, 1908, p. 1859.

THE so-called family diseases form a group which, whether considered from their anatomy or pathological physiology, cannot be mistaken for any other disease. They are the consequence of the original constitution of certain nerve tracts or cells, and so they have sometimes been called Evolution Diseases. They can hardly be termed "Diseases," for they are organic types abnormal from their very origin.

Family diseases are exclusively created by the parents, and they develop quite apart from any external influence. These "family diseases" are often met with as isolated cases, and it is by their own symptoms that they can be recognised.

The clinical types which constitute the family diseases are not found to be identically the same in all cases. Each family tends to work out its own disease. The following classification is proposed for the group "Hereditary Ataxias":—

- a. A spinal form with loss of reflexes, scoliosis and club-foot added to the common syndrome. This would be the Friedreich type.
- b. A cerebellar form with atrophy of the optic nerve, vertigo, mental disturbances, added to the common syndrome forming the Marie type.
- c. A bulbar form with vomiting dyspnoea, and cardiac arrhythmia.
- d. A bulbo-pontine form characterised by auditory disturbances.
- e. A generalised form as in Menzel's case.

These are all different types, but linked up together by the common cerebellar syndrome.

The essential characters of family disease are: (1) they affect in

an identical form several children in the same generation ; (2) they make their appearance at the same age in all of them ; (3) and they originate without the intervention of any morbid agent whether extrinsic or intrinsic.

Of these three fundamental characters the first two may be wanting.

The absence of all known efficient cause is therefore the principal character of family diseases.

The morbid anatomy is characterised by a lesion which consists simply in the disappearance of a system of cells or fibres which dissolve without leaving a trace.

There is a complete absence of any inflammatory reaction.

The course of the disease is always progressive. There is never any retrogression, never does a symptom fade or disappear.

The patient is doomed from the very instant of his conception, as soon as the spermatozoon and ovum have united.

The mechanism by which this premature change takes place is obscure. Is there in each cell a certain dose of energy which gradually gets exhausted till it entirely disappears? Is there a poisoning of the nerve cells through the formation of a special cytolsin? or are the sclerotic changes brought about by an accumulation of waste products, the result of cell activity?

The last hypothesis is rather tempting, for it enables us to understand why it is that the onset of the disease takes place at about the same age in several members of the same family—that is to say, in individuals whose cells have the same chemical characters and the same medium. In the course of development there are thrown into the circulation of the individual at certain periods of his life substances that are very toxic to his nerve cells. The influence of these internal secretions is such that puberty or castration bring about a veritable revolution in the system. Now, is it not chiefly at the time of puberty that the family diseases of the nervous system become manifest? Whether they be brought about in this way or by other means, heredity governs their etiology. Family diseases represent veritable degenerative stigmata in the race. For the purpose of general classification these diseases should be grouped under the following heading:—"Premature physiological senescence of certain organic systems." This physiological senescence is quite independent of any external factor.

F. E. BATTEN.

TYPHOID BACILLI IN THE CEREBRO-SPINAL FLUID IN (517) TYPHOID FEVER. (Ueber die Auffindung der Eberth-Gaffky'schen Bacillen in der Cerebrospinalflüssigkeit bei Typhus abdominalis.) L. SILBERBERG, *Berl. klin. Woch.*, 1908, p. 1354.

SILBERBERG examined the cerebro-spinal fluid of nine typhoid patients. Of these one had a severe attack, five were moderately severe, and three were mild cases. Although seven showed organisms resembling typhoid bacilli, in only the severe case did the culture tests give a positive result. In its physical and clinical characters the cerebro-spinal fluid of these patients did not differ from the normal. Its agglutinative action was very slight, whereas its bactericidal power was much more marked. The removal of the fluid was found to have a beneficial action. The headache in most cases disappeared, and the patients generally appeared brighter.

J. D. ROLLESTON.

TYPHOID MENINGITIS WITHOUT OTHER LESIONS. R. S. (518) LAVENSON, *Univ. of Pennsylvania Med. Bull.*, 1908, p. 55.

A WOMAN, aged 26 years, who was stated to have had typhoid fever several years ago, and had recently been nursing a case of that disease, was admitted to hospital with the symptoms of typhoid fever on April 22nd. Widal's reaction on the 23rd was negative, but on the 26th, the eighth day of disease, was positive. On the 27th lumbar puncture was performed, and a pure culture of typhoid bacilli obtained. Death took place on May 4th. Post mortem no intestinal lesions were found, nor was the spleen enlarged. There was thick pus in the pia-arachnoid covering the convexity of the right hemisphere, and the frontal region of the left convexity showed a similar small focus. In the meningeal exudation organisms were found which resembled morphologically typhoid bacilli.

J. D. ROLLESTON.

PURULENT CEREBRO-SPINAL MENINGITIS CAUSED BY THE (519) TYPHOID BACILLUS WITHOUT THE USUAL INTESTINAL LESIONS OF TYPHOID FEVER. J. NORMAN HENRY and RANDLE C. ROSENBERGER, *Proc. Path. Soc. Philadelphia*, 1908, p. 52.

A MAN, aged 34 years, who had been ill six days, was admitted to hospital with headache, dizziness, constipation and fever. Death took place three days later. Lumbar puncture during life showed a turbid fluid from which typhoid bacilli were isolated. No

meningococci were found. The autopsy revealed purulent cerebro-spinal meningitis, very slight enlargement of the mesenteric glands, and a slight change in Peyer's patches, but no typical typhoid lesions. Sections of the cord and cerebellum showed typhoid bacilli.

J. D. ROLLESTON.

TUBERCULOUS MENINGITIS IN INFANTS. (*La méningite (520) tuberculeuse du nourrisson.*) WILLERVAL, *Thèses de Paris*, 1907-08, No. 452.

THIS thesis, which should be compared with that on the same subject by Clot (*v. Rev. of Neurol. and Psych.*, 1908, p. 291), contains forty-seven observations, six of which have been hitherto unpublished, of tuberculous meningitis in children, whose ages ranged from six and a half months to seven and a half years. The disease is much less rare in infants than is supposed. Four clinical forms are described—eclamptic, hemiplegic, somnolent, and hydrocephalic. The diagnosis is often impossible, and should always rest on the findings of lumbar puncture. The disease may sometimes resemble tetany. In the diagnosis one must also exclude acute non-tuberculous meningitis, which is far from rare in nurslings, and may be due to Pfeiffer's or Weichselbaum's bacilli or *B. coli*.

J. D. ROLLESTON.

A CASE OF GONORRHOEAL MENINGITIS. (*Ein Fall von (521) Meningites gonorrhoeica.*) R. DE JOSSELIN DE JONG, *Centrall. f. Bakt. Originale*, Bd. xlv., 1908, p. 501.

A MALE, aged 19 years, a few weeks after becoming infected with gonorrhoea, presented signs of cerebro-spinal meningitis. There were no symptoms of myelitis or neuritis. The fever was intermittent. The heart and other organs were not affected. The first lumbar puncture showed a slightly turbid fluid in which were many pus corpuscles, but no meningo- nor gonococci. Eight days later the fluid showed, in addition to a number of polymorphonuclears, numerous intra- and extra-cellular diplococci, which their morphological characters and cultural reactions proved to be gonococci. The third puncture, made eight days later, showed a clearer fluid with fewer pus cells and fewer gonococci. Complete recovery took place. The present case, according to De Jong, is the first in which gonococci have been found in the cerebro-spinal fluid, and is an excellent example of a hæmatogenous metastasis of gonococci to the meninges within a few weeks of the primary infection. The therapeutic success of lumbar puncture was striking.

J. D. ROLLESTON.

ACUTE SYPHILITIC MENINGITIS. AN ATTEMPT AT CLASSIFICATION. (522) **FICATION.** (*Méningites syphilitiques aiguës. Essai de classification.*) R. DE COUX, *Thèses de Paris*, 1907-08, No. 445.

THIS thesis, inspired by Boidin, contains twelve cases, four of which have been abstracted in this Review (1907, p. 901, and 1908, pp. 174, 290, and 419). De Coux distinguishes meningitis of the secondary period from that of the tertiary. The former develops early, usually co-exists with the cutaneous eruption, and may, therefore, be regarded as a meningeal enanthem. Its symptoms are those of a diffuse meningitis without localisation phenomena, and resemble those of the ordinary tuberculous meningitis of children. Lymphocytosis is constant, and often considerable. Recovery without sequelæ is the rule. Acute tertiary meningitis is only an episode in the course of a more or less latent chronic syphilitic meningitis, and is due to congestion surrounding the sclero-gummatous lesions. Its phenomena are much more obtrusive than those of secondary meningitis, and consist of violent delirium, convulsions and localisation phenomena, such as epileptiform attacks and palsies of the face and limbs. Death may occur, but more frequently the acute symptoms subside, and a chronic localised meningitis persists. Tertiary meningitis must be distinguished from seizures in general paralysis and from tuberculous meningitis in the adult, uræmia and cerebral tumour. The lymphocytosis which is the rule in chronic syphilitic meningitis may be replaced by an excess of the polymorphonuclears, as in the case of Claisse and Joltrain (*v. Rev. of Neurol.*, 1908, p. 290). The cerebro-spinal fluid may then appear turbid or puriform, but the polymorphonuclears still preserve their morphological integrity. The spirochæta pallida has not been found in either form of meningitis.

J. D. ROLLESTON.

CEREBRAL AND CEREBRO-SPINAL MENINGITIS IN THE COURSE OF THE PUERPERIUM. (523) **COURSE OF THE PUERPERIUM.** (*Méningites Cérébrales et Cérébro-Spinales Suppurées au Cours de la Puerpéralité.*) F. COMMANDEUR, *L'Obstétrique*, June 1908.

COMMANDEUR has collected from the literature fifteen cases of suppurative cerebro-spinal meningitis occurring during pregnancy and in the puerperium; cases of tuberculous meningitis are excluded. The association of meningitis and pregnancy is thus extremely rare, but the author has been able to formulate certain generalisations from the material at his disposal. It is more common in multiparæ than in primiparæ, in the proportion of nine of the

former to three of the latter, the parity not being mentioned in three of the cases. It is more common during pregnancy than in the puerperium. Of the nine affected during pregnancy, five were at full time, one at $8\frac{1}{2}$, one at 8, one at $7\frac{1}{2}$, and one at 7 months. Of the six in which the symptoms were first noticed in the puerperium, two were attacked on the day following confinement, one on the second day, one on the third, one on the sixth, and one not till four weeks after delivery. In only one case was the meningitis secondary to a puerperal infection, and in it the organism present was a staphylococcus. Two cases were secondary to pneumonia, one to pneumococcal pleurisy, and one to suppurative otitis. In the remaining ten the condition was apparently primary, and in them the organism was usually the pneumococcus—in fact, the bacteriological findings did not differ from those obtaining in cerebro-spinal meningitis apart from pregnancy. The chief interest of the condition when occurring in the pregnant or puerperal woman lies in the possibility of mistaking it for eclampsia, a diagnosis which was made in several of the recorded cases, and only rectified on post-mortem examination. The two conditions ought never to be confounded if a thorough examination is made. The headache in cases of meningitis is more severe and continuous than in eclampsia, the convulsions more localised, and the coma deeper and occurring only as a terminal phenomenon. Kernig's sign may or may not be present. Albuminuria is usually present in both conditions. All doubt is removed if the spinal fluid is examined by means of spinal puncture. Treatment resolves itself into an attempt to save the child. All the mothers died, but in all but one case the child remained alive till the death of the mother. Two of the nine cases died undelivered, but in one a post-mortem Cæsarean section was unsuccessful in saving the child. In four labour occurred spontaneously during the illness, and living children were born. In three delivery was effected instrumentally, in two the mother being *in extremis* at the time of interference; all the children survived. In all cases of cerebro-spinal meningitis in pregnant women an attempt ought to be made to save the child.

B. P. WATSON.

**ANALYSIS OF 400 CASES OF EPIDEMIC MENINGITIS TREATED
(524) WITH ANTIMENINGITIS SERUM. FLEXNER and JOBLING,
Journ. of Am. Med. Assoc., July 25, 1908.**

DRS FLEXNER and JOBLING have already published elsewhere (*Journal of Experimental Medicine*, Jan. 1908) the first series of cases treated with their serum. They now give statistics of the 393 cases treated up to date. These have been collected from the

different centres in Great Britain and America in which the serum has been tried. They exclude cases which terminated fatally within twenty-four hours of the first injection, on the ground that such a period is too short for any influence to have been exerted by the treatment. In such a disease as epidemic cerebro-spinal fever, however, these cases might with advantage have been included, as in every outbreak a certain number of fulminant cases are almost sure to occur, and this type of the disease may run its whole course in twelve hours. To omit such cases, then, destroys to some extent the value of the comparison of the serum death-rate with that of the disease treated on ordinary lines. Of the 393 patients included in their tabulation, 98, or only 25 per cent., died. We may remark here that, without deductions, the mortality is only 30 per cent., a most remarkable figure.

The disease was most fatal in infants of under one year, of whom 50 per cent. succumbed, and in adults of over twenty years of age. In 328 of the cases it was possible to determine with reasonable accuracy the exact day of the onset of the illness. From these it was found that the mortality of patients who came under treatment from the first to the third day after the first symptom was only 14·9 per cent. Of the patients who received their first injection from the fourth to the seventh day, 22 per cent. died, and thereafter the mortality rose to 36·4 per cent. These figures show very strikingly the advantages of early injection, and also emphasise the value of the serum itself. Not a few patients in the third group were chronic when first injected, and the outlook for cases of this type is not wholly discouraging. Indeed, so long as diplococci are present in the exudate, and mechanical damage to structure is not irreparable, it is well worth while to administer serum injections. From 25 to 30 per cent. of the cases terminated by what might be fairly termed a crisis.

As regards the result of the injections on the diplococci themselves, they are rapidly reduced in numbers, become wholly intracellular, and show swelling and fragmentation. Coincidentally they lose viability in cultures. As to the spinal fluid, turbidity soon disappears, and even purulent exudates occasionally clear up quickly. After injection a very rapid or even critical fall of leucocytes often takes place. Progressive increase of turbidity, rise of leucocytosis, and persistence of diplococci in the fluid are bad signs. Relapses are infrequent, and, if vigorously treated with serum, seldom fatal. Generally speaking the number of complications has been small. The only persistent defect noted in the series of cases was deafness, and this was not common, and, when observed, had often been present to some degree before the treatment was commenced.

CLAUDE B. KER.

THE EPIDEMIOLOGY OF ACUTE POLIOMYELITIS. L. EMMETT
(525) HOLT and FREDERIC H. BARTLETT, *Amer. Journ. of the Med. Sci.*,
May 1908.

THIS is a very interesting analysis of thirty-five epidemics of acute poliomyelitis collected from the literature. The original references have been consulted in all but one instance. The most important epidemics are those which occurred in Norway and Sweden in 1893, and in New York during the past year. No less than five epidemics have been reported in Norway during the last ten years.

All but two of the thirty-five epidemics above referred to were confined to the hot season only. The most frequent months were July, August and September, the epidemic terminating in almost every case with the month of October.

Contrary to what is so often seen after epidemics of cerebral spinal meningitis, in but one instance is it stated that there was an increase in the number of sporadic cases in succeeding seasons after an outbreak of poliomyelitis. The epidemics have been fairly equally divided between city and country. In the majority of instances the hygienic surroundings were good. Only one example of the occurrence of an epidemic in a crowded community has been reported.

No association with other diseases can be said to have been established. On the contrary the great majority of patients are stated to have been well at the time of onset. Severe cholera infantum prevailed concurrently with the Stockholm epidemic of 1895. While both cerebro-spinal meningitis and gastro-enteritis prevailed in Vienna in 1897, during the epidemic of poliomyelitis of 1898 these diseases were not common. Landry's paralysis, acute encephalitis and multiple neuritis had been described in a few epidemics. In all probability these were cases of poliomyelitis erroneously diagnosed.

The mortality in 1659 cases collected from different records of epidemics in which this point is specially referred to was 12.1.

Observations bearing upon the communicability of the disease may be considered under three heads: (*a*) the general distribution; (*b*) the occurrence of more than one case in a family or household, and the interval between the onset in the different cases; (*c*) the development of poliomyelitis after contact with a person suffering from the disease in an acute stage, or after moving into a district where the disease is prevailing.

In the majority of epidemics there have been a comparatively small number of cases, and these have been widely distributed. The authors have collated and tabulated forty instances of more than one case in a household. Among these there were thirty-seven in which the second case developed within ten days and

thirty-three in less than a week, while in thirteen the interval was less than twenty-four hours. Pasteur reports seven cases in one family. In another family four cases occurred. Two sisters were taken ill within six days of each other, the father seven days later, a son two days after the father's death. There are five groups of three cases in a family, while instances of two are comparatively common. Dr Holt has met with three examples which he describes as follows:—

“A boy aged 14 months was taken ill on the 23rd of the month and died on the 27th with bulbar symptoms. A sister aged 5 years was taken ill on the 26th, and recovered with paralysis of both lower extremities. No other cases are known to have occurred in the town at the time.”

“During the epidemic of 1907, in a family living in a village near New York, a boy aged 8 years was taken ill on Sunday and died on the following Sunday morning. His brother, aged 5 years, was attacked the day before the death of the older boy, and recovered with paralysis. No other children in the family. Only one other case in the village, living more than a mile away.”

“There were admitted to the Babies' Hospital in October 1907 two children aged about 2½ years from a country home containing twenty young children. The children attacked with poliomyelitis occupied adjoining beds in the institution. The second child was taken ill seven days after the first one. There were no other cases in the house.”

In one epidemic of seventeen cases referred to by the authors a group of ten occurred in the same neighbourhood; two cases were in the same house; two others were sisters; two others were brothers; the other four children attacked lived near by. In the second group there were seven cases in the same neighbourhood; three occurred in the same family; a fourth lived in an adjoining house and was attacked just ten days after he came there to reside; the other three children lived close by.

Among instances of cases occurring after moving into a district where the disease is prevalent were the following:—Two children (observed by Dr Holt) were taken ill simultaneously ten days after their return to New York during the epidemic of 1907. Another case was that of an infant aged nine months who was put into the same crib with a child suffering from poliomyelitis. Within eight days (the exact time was not determined) the child was affected with fever followed by paralysis. Again, three servant girls living in different houses returned together to their home. While there and shortly after their arrival all three became ill with acute poliomyelitis.

The following are the conclusions arrived at by the authors:—
“The occurrence of epidemics and the relation of certain groups of

cases to one another in these epidemics place beyond question the statement that acute poliomyelitis is an infectious disease. Whether we can go farther and state that the disease is communicable is an open question. After carefully considering all the evidence brought together in these papers, we cannot resist the conclusion that the disease is communicable, although only to a very slight degree, one of the most striking facts being the development of the second cases within ten days after possible exposure. Positive statements, however, must be deferred until the discovery of the infectious agent."

A table is appended which gives the principal facts regarding the epidemics collected, together with the references to the literature.

EDWIN BRAMWELL

THE OCCURRENCE OF INFANTILE PARALYSIS IN MASSACHUSETTS IN 1907. R. W. LOVETT, *Boston Med. and Surg. Journ.*, July 30, 1908, p. 131.

THIS is a report prepared for the Massachusetts State Board of Health, with special reference to the etiology of the disease. It deals with some 234 cases, and the evidence as to the etiology is summed up as follows:—The character of the onset suggests infection, but the case is not proven bacteriologically. But negative evidence does not disprove the theory of infection, for the organism present, if one exists, may liberate a toxin and disappear. The seasonal occurrence, the age incidence, and the frequent association of intestinal disturbance with the onset suggest some intestinal infection. It seems reasonable to suspect that some bacillus, probably an anaërobic one, reaches the intestines in milk, and there liberates a toxina which is absorbed and is carried to the spinal cells in the blood current. But it cannot be asserted that the disease is always due to the same organism, or even that it is a pathological entity. It may be simply the clinical expression of the reaction of the spinal cord to one of several causes, of which infection may well be one. The data are not sufficient to establish the idea of contagion, but the distribution, spread, etc., warrant us suspecting it, and isolation of children during the acute attack is recommended.

J. H. HARVEY PIRIE

EPIDEMIC INFANTILE PARALYSIS. M. ALLEN STARR, *Journ. (527) Amer. Med. Assoc.*, July 11, 1908, p. 112.

THIS is an account of an epidemic occurring in New York and neighbourhood in the summer of 1907, affecting about 2000 cases. Lasting from May to December, it was at its height in August

and September. In few of the cases was there any preceding infectious disease. Excessive sweating was found a common symptom in the early stages. Paralysis was generally noticed on the third or fourth day. The type was sometimes the ordinary poliomyelitis, in others with accompanying bulbar paralysis, in others, again, with polioencephalitis. Pain was a usual symptom. Anaesthesias were not observed. Complete recovery occurred in many cases, but there was a mortality of about 7 per cent. No definite causal agent was detected. Urotropin was found useful in the early stages, the liberated formaldehyde being found in the cerebro-spinal fluid. When the pain has subsided the use of strychnine is recommended—to be pushed as far as is consistent with safety. Orthopedic treatment for the prevention of deformities is of the utmost importance. A short account of forty-four other epidemics is included in the article.

J. H. HARVEY PIRIE.

**ACUTE ANTERIOR POLIOMYELITIS IN THE ADULT, WITH
(528) EXHIBITION OF A CASE.** LA SALLE ARCHAMBAULT, *N.Y.*
Med. Journ., Aug. 8, 1908, p. 255.

THE case was that of a man of forty, who, after four days of fever, headache, and vomiting, became paralysed in the left arm. In three days all four extremities were involved, especially the left arm and right leg. The trunk and neck muscles were affected, but not those of the face. At the onset pain was a marked feature along the spine and in the lower limbs. There was slight temporary retention of urine. There were no sensory disturbances save slight generalised hyperalgesia, and there was both subjective and objective coldness, affecting especially the right lower limb. The case would appear to be one of anterior poliomyelitis associated with some degree of meningitis. Lumbar puncture had not been done at the time of exhibition of the patient.

J. H. HARVEY PIRIE.

THE OCULAR MANIFESTATIONS OF TABES DORSALIS.
(529) (*L'œil tabétique.*) MASSIA and DELACHANAL, *Gaz. des Hop.*,
Jan. 1908, p. 3.

THIS is the first instalment of a paper upon the ocular troubles in tabes. The motor troubles are discussed fully, special emphasis being laid on the frequency of the external ocular paralyses, and upon their occurrence with different clinical features in the prætaxic and in the fully-developed stages of the disease. The prætaxic palsies are distinguished by being monocular, isolated,

rapid in their onset, by affecting branches of different nerves, and by being fleeting and benign in their course. They give a valuable clue to the diagnosis, especially in luetic subjects with few other signs. The later paralyses are less frequent, often bilateral, affect the third and sixth nerves too, and often all the branches of the former. Their onset is slow, they gradually get worse, they persist.

Diplopia is fully discussed, as regards signs, symptoms, and diagnosis, also ptosis and strabismus. Under the pathogenesis of the transitory palsies, the reflex, vascular, and neuritic theories are noted, and Brissaud's idea of their being, like the later ones, due to central lesions. The relationship between tabes and exophthalmic goitre is emphasised. Inco-ordination of the eye muscles, comparable to that of the limbs, seems to be not so uncommon, although nystagmus is almost limited to the ataxia of Friedreich.

Myosis, estimated as present in 50 per cent. of cases, is very rarely accompanied by external ocular paralysis, while accommodative paralysis and papillary atrophy seldom accompany myosis. The Argyll-Robertson pupil is its only concomitant. Mydriasis, less frequent, usually goes with optic atrophy with defective vision, although rarely myosis is then present. Convergence and accommodative pupillary contraction seem variously influenced, but they are usually absent in mydriasis. Mydriasis is possible without ophthalmoplegia, presumably from stimulation of the sympathetic. An average of 30 per cent. have neither myosis nor mydriasis.

In anisocoria the abnormal pupil is the one which reacts less well to light. Both eyes must be equally illuminated. In physiological anisocoria (which, by the way, Fuchs does not acknowledge) both pupils react equally well to light. The various causes of dilatation and contraction are given fully, and the differential diagnosis.

Pupillary irregularity, usually in mydriasis, unilateral at first but bilateral later, is often a very early sign. Joffroy says that all Argyll-Robertson pupils show irregularity. The pupils may undergo transitory alterations in size and shape before the gastric crises and during the lightning pains.

Atrophy of the iris, evidenced by the loss of the markings and ridges—radial in the pupillary zone and concentric in the peripheral zone—is often seen. It may be local or general, and depends upon the state of the ciliary ganglion.

Accommodation is frequently affected, either by spasm—with its accompanying photophobia, lacrymation, and even dissociation between convergence and accommodation—or by partial or complete paralysis, due to third or ciliary nerve lesions.

The pupil reflexes and the sensory and trophic affections are to be dealt with in the subsequent instalments of their review.

W. CLARK SOUTER.

TABES ASSOCIATED WITH TROPHIC CHANGES SUGGESTING (530) ACROMEGALY. F. X. DERCUM, *Journ. of Nerv. and Ment. Dis.*, Aug. 1908, p. 507.

THIS is a typical case of tabes, but the patient presented in addition certain trophic changes suggestive of acromegaly. Six years after the commencement of the tabes, and two months before the patient's death, it was noticed that his face had become lengthened, that his chin was protruding, that his nose was prominent, as were also the zygomatic arches and the occipital protuberance. The hands and wrists were also large, the heels were much enlarged, as were the internal condyles of the knees. The joints were apparently normal. The acromial ends of the clavicle were enlarged, the ribs were broad and thickened, and there was some spinal curvature in the dorsal region.

At the autopsy tuberculosis of the lungs, chronic myocarditis and interstitial nephritis were present. The hypophysis was twice as large as normal. Its capillaries were engorged with blood. Some of the cells were fragmented and apparently degenerated. The suprarenal glands were somewhat enlarged, but no marked changes were noticed save that the cells of the zona fasciculata seemed to have undergone an unusual degree of fatty change. There was a marked sclerosis in the posterior column of the cord.

The author concludes by remarking, "It is not improbable that the changes in the pituitary body bear some relation to the bony changes. The thought suggests itself that perhaps in cases of tabes generally, where there are marked trophic changes in the bones there are also changes of the pituitary body, and it may be wise to examine the pituitary body and the other ductless glands in such cases."

EDWIN BRAMWELL.

PRURITUS IN TABES. (Du prurit tabétique avec ou sans lichénification.) J. REBAUD, *Thèses de Paris*, 1907-08, No. 287.

THIS thesis, inspired by Milian, who first described this symptom (*v. Rev. of Neurol. and Psych.*, 1907, p. 909), contains thirty-nine cases. Of twenty-seven personal cases of tabes eight gave a history of pruritus. Among these, thoracic pruritus was most frequent. There was only one case of pruritus ani and not a single case of lichenification.

J. D. ROLLESTON.

CLINICAL AND ANATOMICAL OBSERVATIONS UPON SIX (532) CASES OF PSEUDOSYSTEMIC DISEASE OF THE SPINAL CORD, etc. (Klinische und anatomische Untersuchungen von sechs Fälle von Pseudosystemerkrankung des Rückenmarks: Kritik der Leben von den Systemerkrankungen des Rückenmarks.) NONNE und FRÜND, *Deutsche Zeitsch. f. Nervenheilk.*, Bd. 36, S. 102, 1908.

SYSTEM diseases of the cord may be divided into two main classes, hereditary and acquired. The hereditary group comprises Friedreich's ataxia and the hereditary spastic spinal paralysis of Strümpell, Bernhardt, and others. The acquired forms are much more varied and numerous, including tabes and tabes-paralysis, pellagra, and the degenerations occurring in such diseases as pernicious anæmia, carcinoma, and chronic alcoholism. These again may be subdivided into the true system diseases, spastic or ataxic, or a combination of spastic and ataxic; and lastly, the pseudo-system diseases of several regions of the cord. Nonne and Fründ here discuss the question whether any sharp distinction can really be drawn between the true system diseases and the pseudo-system diseases, ultimately answering this question in the negative. Tabes, which is a disease *sui generis*, is expressly excluded from the group of diseases here discussed.

CASE 1.—The clinical notes have been lost. The following conditions were present in the spinal cord:—In the posterior columns, symmetrical degeneration of Goll's and Burdach's tracts in the lumbar region, with normal fibres in the septo-marginal area. In the dorsal region, total degeneration of the posterior columns, except a small area close to the posterior cornua and at the periphery of Goll's tract. A similar condition extended up to the upper cervical region. In the lateral columns, symmetrical degeneration of the lateral pyramidal tracts in the lumbar region; in the dorsal and cervical regions, degeneration of the lateral pyramidal and dorsal cerebellar tracts; in the anterior columns, unilateral degeneration of the ventral pyramidal column in the lower dorsal region, bilateral degeneration in the upper dorsal region, normal appearance in the cervical region.

CASE 2.—A male patient of 56 years, free from hereditary taint, who had severe gastro-intestinal disorder three years before the onset of his illness. No syphilis, but alcoholic excess for three years. No pains or paræsthesia. Flaccid paresis of the legs, with loss of the deep reflexes and extensor plantar reflexes. Towards the end, weakness and ataxy of the arms, and distinct sensory disturbances. Bladder and rectum early affected. Pupils normal. Febrile enteritis with recurring exacerbations. Severe anæmia.

Microscopically the cord showed foci of sclerosis in the posterior columns in the lumbar region; systematic degeneration of Goll's tracts in the dorsal and cervical region; in addition, sclerotic foci in Burdach's columns in the cervical region. The lateral pyramidal tracts showed sclerosis, increasing in intensity from the lumbar region upwards. The dorsal cerebellar tracts were sclerosed in their whole extent. Degeneration of the ventral pyramidal tracts began below in the mid-dorsal region, overlapping the ventral pyramidal tract in the upper dorsal region, and could no longer be recognised in the upper cervical region. The anterior and posterior cornua were normal, save for the cells of Clarke's column, which were atrophic. Anterior and posterior roots normal.

The disease lasted ten months, with a probable earlier attack three years previously.

CASE 3.—Two years before the beginning of the disease the patient, a man of 36, had severe anæmia with general glandular enlargement, followed by iritis. For a year and a half the patient felt well, then began to have weakness and spasticity of the legs. Syphilis and alcohol were denied. The pupils were normal. The gait was spastic-ataxic, with increased deep reflexes. No sensory changes at first. Bladder normal; later, the bladder became paralysed, and there were severe pains and girdle sensations. Moderate anæmia. Duration of disease, eight months.

Anatomically: in the posterior column commencing degeneration in the lumbar region of focal character, nearly symmetrical. In the dorsal region, symmetrical degeneration of the greater part of Burdach's and Goll's tracts. In the cervical region, symmetrical degeneration of Goll's tracts; new foci in Burdach's tracts. The lateral pyramidal tracts showed symmetrical degeneration beginning in the lumbar region, extending to the upper dorsal region, where it was more diffuse. In the cervical region marked restitution took place around the grey substance. Isolated empty areas in the medulla oblongata. The ventral pyramidal tracts were diseased, commencing with empty areas in the lumbar region. In the dorsal region both ventral pyramidal tracts were totally degenerated, overflowing to the periphery of the cord anteriorly. In the cervical region there was a distinct lessening of degeneration. The dorsal cerebellar tracts were sharply and systematically diseased from the dorsal region upwards, and this could be traced into the medulla. The cells of Clarke's column were rarified. In the diseased areas marked vascular changes were present.

CASE 4.—Forty-one year man, with negative family and personal history. No venereal disease, but moderate alcoholism. Began with pains and weakness of the legs. At first the paresis was spastic-ataxic, with increased deep reflexes and extensor plantar response. Sensory losses were well marked. Microscopically

there were focal degenerations in the posterior column in the lumbar region, which degeneration in the dorsal region included the whole space between the posterior horns. In the cervical region there was systematic degeneration of Goll's column and new sclerotic foci in Burdach's column. In the lumbar region there was marginal degeneration and thickening all round the periphery. The degeneration of the lateral pyramidal tracts began in the lower lumbar region, and in the dorsal region over-spread beyond the boundary of the pyramidal system. In the cervical region healthy fibres entered the tract from the grey matter. Total degeneration of the dorsal cerebellar tracts, forming sclerotic areas in the lower part; in the upper part showing empty fibres. The cells of Clarke's column were diminished. Diffuse degeneration of both ventral pyramidal tracts in the whole of the dorsal and cervical regions.

CASE 5.—A man of 50 years, whose mother died of some nervous malady, but who himself had an excellent previous history, free from alcoholic or venereal taint. The illness began with gastro-intestinal symptoms. Then he developed feeling of weakness and stiffness in the legs. The upper extremities had spasms and intention-tremors, the legs were paretic and spastic, the deep reflexes were increased, with extensor plantar reflexes. Sensory blunting was also present in the legs. The spastic paresis ultimately became flaccid. Post-mortem, there were focal degenerations in the posterior column of the lumbar region, with a pure sclerosis of Goll's column in the dorsal and cervical regions. Focal degeneration also in Burdach's column in the dorsal and lower cervical regions. In the lumbar region there was commencing degeneration of the lateral pyramidal tracts, overstepping the limits of the tract itself, but less intense in the cervical region. The central parts of the cord were also sclerosed. The dorsal cerebellar tracts were completely degenerated. There was irregular degeneration of the ventral pyramidal tracts in the dorsal and cervical regions. The anterior and posterior grey matter were normal, but the cells of Clarke's column were atrophied. The total duration of the illness was eight months. There was moderate anæmia of simple type.

CASE 6.—A man of 47 years, without hereditary taint, had severe anæmia two years before the onset of his later symptoms, together with transient paræsthesias, sensory change, and absent knee-jerk. A second attack occurred, without anæmia, beginning with flaccid paresis and ataxia, without sensory loss. Later the paresis became spastic, sensory change reappeared, also loss of control of the sphincters.

In the posterior column there were foci of sclerosis in the lumbar region. In the lower dorsal region the whole of the

posterior columns were degenerated. In the upper dorsal and in the cervical region there was symmetrical degeneration of Goll's and partly of Burdach's tracts, with isolated small foci in addition. The pyramidal tracts and the whole anterior columns were sclerosed from the upper lumbar region upwards, diminishing in intensity in the cervical region. Anterior and posterior cornua and roots were normal.

All the above cases are examples of combined system-disease of the cord. In three out of the six the spinal affection was preceded by a severe febrile malady, at periods varying from one and a half to two and a half years before. Two cases had severe anaemia. It was noticeable that no two cases were exactly similar, either clinically or anatomically. But, on the whole, it was evident that the posterior columns were most severely affected. The pyramidal system was also constantly degenerated, the other parts of the lateral column being only sclerosed when the marginal zone was diseased. The affection of the anterior column was most irregular in form and extent, though the ventral pyramidal tracts were often systematically affected. The grey substance and its surrounding white matter seemed to be relatively less affected. This was apparently associated with the normal condition of the vessels of the grey matter.

To sum up, the authors conclude that there is no real combined "system" disease, in the strict meaning of the term; moreover, the slightness of the clinical symptoms is often in marked contrast to the severity of the anatomical change. The degeneration is not limited to strict systems or tracts; there are present, in addition, diffuse non-systematic degeneration. In acute cases this marked process appears to commence as small-cell infiltration around the blood vessels.

Finally, the authors point out that true system degenerations are best exemplified, not by cases such as are above described, but by cases of family and hereditary spinal diseases, amyotrophic lateral sclerosis, and tabes dorsalis, with its various combinations.

PURVES STEWART.

UNILATERAL CAUDA EQUINA SYNDROME. (*Syndrome de (533) l' "hémi-queue de cheval" par meningo-radiculite syphilitique.*)

LAIGNEL-LAVASTINE, *Nouv. Icon. de la Salpêtrière*, March-April 1908, p. 117.

THIS is an interesting case of involvement of the right fifth lumbar and all the right sacral roots in a mass of syphilitic meningitis or "meningo-radiculitis" at the level of the exit of the third lumbar

root, with a typical clinical picture. The case is of value inasmuch as the pathological examination demonstrates accurately the difference between the exogenous and the endogenous fibres at these levels. According to Laignel-Lavastine, his case supports Nageotte's contention that Lissauer's zone is composed of fine vertical endogenous fibres.

S. A. K. WILSON.

A CASE OF BULBO-PONTINE SOFTENING. (*Étude Anatomoclinique d'un cas de ramollissement bulbo-protubérantiel.*)

FRANÇAIS and JACQUES, *Rev. Neurol.*, June 15, 1908, p. 521.

CLINICALLY: inability to swallow, absence of the pharyngeal reflex, paralysis of the left side of the palate, weakness of the left limbs, inability to stand alone, tendency to fall to the left, slight general ataxia, dissociated anæsthesia on the right side, including the face, myosis and narrowing of the palpebral fissure on the left side, a little nystagmus to both sides, double flexor response. Pathologically: three limited areas of softening, one dorsal to the left inferior olive, destroying the ventral cerebellar tract, the nucleus ambiguus, the greater part of the grey reticular formation, the upper part of the sensory decussation, some of the fibres of the bulbar part of the spinal accessory, the descending sensory root of the fifth, the ventral part of the corpus restiforme, the superficial arcuate fibres and many cerebello-olivary fibres; a second towards the middle part of the pons on the right side, involving the right pyramidal tract, and some of the transverse fibres of the middle cerebellar peduncle; and a small third area at the same level, invading the outer end of the left fillet.

S. A. K. WILSON.

CONTRIBUTION TO THE DIAGNOSIS AND TREATMENT OF
(535) **TUMOURS OF THE CENTRAL NERVOUS SYSTEM.**

(*Beiträge zur Diagnostik und Therapie der Geschwülste im Bereich des Zentralen Nervensystems.*) H. OPPENHEIM.
Sn. 193. Mit 20 Abbildungen im Text und 6 Tafeln. Verlag von S. Karger, Berlin, 1907.

STATISTICS collected from the literature must give, it is obvious, a quite erroneous impression of the results obtained in operations for the removal of intracranial tumours. We have in the present monograph a most important contribution to the surgery of tumours of the central nervous system, for the work embodies the personal experience of one man, and he one of the greatest authorities of the day, working in conjunction with surgeons who have had a special experience in this department of surgery.

Oppenheim, in the second edition of his monograph on intracranial tumours (published in 1902), recorded his experience of the results of operation up to that date. At that time, of twenty-four cases which he had submitted to operation, in only one was a cure effected. In 20 to 21 per cent. there was improvement, while in 37 to 38 per cent. death followed as the result of the operation. Of these twenty-four cases, in four a palliative operation only was undertaken.

The present communication embodies the author's experience since 1903. Among twenty-seven cases operated upon, in twenty-four the operation was undertaken in the hope of being able to remove the growth. Three cases (11·1 per cent.) were cured, six (22·2 per cent.) improved, a part of the tumour or a cyst in its substance being evacuated, while in 55 per cent. death occurred immediately, or soon after the operation. This large mortality Oppenheim accounts for by the circumstance that in twelve cases the tumour was situated in the posterior fossa. The conclusion he arrives at from his own material is, that of every nine or ten cases carefully selected and for the most part (grösstenteils) correctly diagnosed, in only one is complete removal and recovery to be expected. All these cases above referred to are recorded in detail.

The paper also includes the records of the cases of tumour of the spinal cord which the author has submitted to operation. These are fifteen in number.

Oppenheim holds that surgical treatment is urgently indicated in any case which presents the typical symptomatology of a tumour of the spinal membranes. According to his experience, in about 50 per cent. of these cases a cure is to be anticipated, and it will be the more complete the earlier the interference is undertaken. More extended surgical experience will doubtless reduce the mortality from shock, and especially from meningitis, which is at present considerable. The typical symptoms of an extra-medullary tumour may be simulated by disease of the vertebræ, by a localised meningeal process, or by a new formation within the spinal cord. As Nonne's material demonstrates, the differential diagnosis between an extra-medullary tumour on the one hand, and a tumour of the vertebral column on the other, is not always possible. Again, the symptomatology of tumours of the spinal membranes is very often atypical, and if we are to reserve operation only for those cases in which the symptoms are practically certain, a considerable proportion of tumours will be missed. If after consideration in a doubtful case the disease is observed to be distinctly progressive and the existence of an extra-medullary tumour appears to be at all probable, an exploratory laminectomy should be undertaken. Where the general diagnosis is uncertain

the diagnosis of the level of the lesion should be as absolute as possible. The operator should not stop on exposing the dura if no tumour is met with, for in only some of Oppenheim's cases of intradural tumour was the new growth visible or palpable before the membrane was opened. That an exploratory laminectomy leads to no harmful result when an intramedullary lesion exists is demonstrated by several cases here recorded, as well as by the observations of others.

EDWIN BRAMWELL.

**A BRAIN TUMOUR LOCALISED AND COMPLETELY REMOVED,
(536) WITH SOME DISCUSSION OF THE SYMPTOMATOLOGY
OF LESIONS VARIOUSLY DISTRIBUTED IN THE
PARIETAL LOBE.** C. K. MILLS and C. H. FRAZIER, *Journ.
of Nerv. and Ment. Dis.*, Aug. 1908, p. 481.

THE case is that of a female aged 45, who suffered from attacks of vomiting, dizziness and headache. When seen by Dr Mills on December 30, 1907, in addition to optic neuritis and left lateral hemianopia, previously detected by Dr de Schweinitz, there was a moderate degree of hypæsthesia of the left extremities together with hypastereognosis, some loss of sense of position, and some ataxia and atactic tremor of the left arm. There was also some impairment of the ability to use her left leg and arm, "this being due to inco-ordination rather than to loss of strength." There was no aphasia. The tendon jerks were normal and the Babinski sign was not present. On February 1st the left pupil was slightly greater than the right, and the associated upward movement of the eyes seemed to be limited but was not lost. Ever since her first symptoms were noticed in October the patient had had a peculiar puffing sound in the left ear and a singing or buzzing sound in the right, although her hearing had not changed. Memory and attention were good. Slight left facial weakness. A little awkwardness in some of the finer movements with the left hand. Sense of location impaired over left upper extremity. Distinct ataxia in finger nose test on left side. Slight hypastereognosis on left side.

On February 3rd it was noted that the swelling of the optic discs had increased from $2\frac{1}{2}$ to 5 dioptries. Wernicke's symptom was not present.

Dr Frazier operated on February 5th. The incision was made so as to expose portions of the occipital, parietal and temporal lobes. A cyst was removed which was found subsequently to measure 8 by 4 centimetres. The cyst came to the surface over an area which did not exceed that of a 25 cent piece in the superior posterior angle of the opening. The operation, which

was performed in one stage, the bone flap being replaced, occupied thirty minutes exclusive of the time taken in closing the wound in the dura and scalp. The cyst proved to be a simple serous cyst.

On February 12 the ataxia had disappeared and all the functions of the arm and leg were restored. There was still five dioptries of swelling of the optic discs. On March 9 the hemianopsia was improving, and on May 8 she had a practically full field on the right side with partial hemianopsia on the left.

The authors remark that the autopsy method of determining the operability of tumours is open to serious objection, and the percentage of tumours suitable for radical operation should be made from the statistics of the surgical clinic and not of the pathological laboratory.

They have studied four different symptom complexes according to the degree of implication of the various portions of the parietal lobe, and they give directions for exposure of the implicated region in each case. The symptom complexes are (1) pronounced hemianopsia and ataxia, combined with pressure symptoms varying in intensity, such as hypæsthesia, hypastereognosis and slight paresis of the face and limbs; (2) astereognosis and ataxia, combined with symptoms showing various degrees of involvement of cutaneous, muscular and arthridal sensibility, but without hemianopsia and with no, or only slight, paresis; (3) hemianopsia and hemiataxia, with hypæsthesia and hypastereognosis and pronounced paresis, specially of the face and upper extremity; (4) astereognosis and ataxia, with hypæsthesia and pronounced paralysis, specially in the lower and upper extremities.

EDWIN BRAMWELL.

A CONTRIBUTION TO THE SYMPTOMATOLOGY OF PARALYSIS

(537) **AGITANS.** (*Ein Beitrag zur Symptomatologie der Paralysis Agitans.*) A. PELZ, *Neurol. Centralbl.*, Aug. 1, 1908, S. 27.

THE case is that of a man aged 59, who presented the characteristic postures of paralysis agitans. While the patient was at rest there was no tremor. Upon movement, however, a tremor was present. The author, after referring to paralysis agitans without tremor, alludes to cases in the literature in which tremor was present only during voluntary movement.

EDWIN BRAMWELL.

THE REVISION OF HYSTERIA. (La révision de l'hystérie.)
(538) HENRY MEIGE, *Pressé méd.*, juillet 4, 1908.

THIS paper formed the basis of a discussion on the subject of hysteria at the meeting of the Neurological Society of Paris.

M. Meige commences by pointing out the necessity of having instituted a discussion for the revision and elucidation of a condition so variable and ambiguous.

He gives us a clear insight into the resultant opinion arrived at by numerous trustworthy observers.

The elusive manifestations of hysteria which have heretofore been regarded as more or less pathognomonic of the condition, and upon which so much stress has been laid, have now been interpreted in such a manner as to lose their significance and prestige.

Among the characteristic and formerly accepted signs of hysteria the following are discussed :—

Hemi-anæsthesia, anæsthesia of the pharynx (or, as he terms it, abolition of the pharyngeal reflex,"), retraction of the visual field, monocular polyopia and dyschromatopsia.

These have been termed the "feigned" stigmata of hysteria. He quotes the opinion formulated by Babinski, Ballet, Brissaud, Dupre, and Souques as being that "the feigned stigmata of hysteria result only from an unconscious suggestion which is usually of medical origin."

Each of these observers cites cases in which one or other stigma was unnoticed by patient or physician on admission or at the onset of the condition, but developed subsequently after examination.

Babinski asserts that the anæsthesia arises from faulty or indiscreet methods of examination, and is the result of suggestion. On these grounds one should never ask the hysterical patient questions like the following :—

"Do you feel that?" "Do you feel as well on one side as the other?" as they suggest to the patient an impairment or absence of cutaneous sensations.

Babinski, after personally examining a hundred cases of hysteria with care, was unable to find hemi-anæsthesia in a single case.

Dejerine, Raymond, and Pitres, recognising that anæsthesia is often of medical origin, maintain that they appear spontaneously in some cases, suggestion taking no part in their genesis.

M. Thomas contends rightly that it is so difficult to prove whether or not any previous or anterior suggestion has been put forward that he finds it impossible to maintain that it has not existed.

With regard to contraction of the visual fields, Rochon-Duvigne and Brissaud, like Babinski, arrive at the conclusion that this disturbance is due to errors in examination.

Dyschromatopsia has also been proved to be absent by Brissaud by adopting the use of the Remy's diploscope, which renders deception impossible on the part of the patient.

Babinski also maintains that abolition of the pharyngeal reflex varies according to the subject, and may be suppressed by patients owing to some suggestion.

The above signs are discussed under the title of "Stigmata of Hysteria."

Next "Les troubles pithiatiques" (disorders curable by persuasion) and so-called hysterical dystrophies are discussed.

Babinski suggests that this group, in which we find convulsive attacks, paralyses, contractures, anæsthesiæ, hyper-æsthesiæ, disturbance of the senses and speech, etc., ought to be recognisable from the fact that they can be produced by suggestion and disappear under the influence of suggestion or persuasion alone.

Raymond, Pitres, and Dejerine think that one ought to regard as hysterical certain circulatory and trophic disturbances, *e.g.* dermatographia, œdema, eruptions, hæmorrhages, ulcerations, etc.

Brissaud is convinced that œdema and ulceration of a hysterical nature do not exist. He cites the case of a young girl who developed a series of abscesses which were considered hysterical but were found each to contain a fragment of needle, presumably introduced by the patient herself.

As regards the bullæ of pemphigus, M. Raymond clearly recognises that such symptoms cannot be produced by suggestive influence, and states that the time is not far distant when another "vaso-motor neurosis" will be proved responsible for these circulatory disturbances; and it is from future investigation that we must learn what the link is which unites this neurosis to the "pithiatique" symptoms, the only indisputable phenomena of hysteria. Thus we see that the vaso-motor or trophic disturbances formerly attributed to hysteria do not appear in the same way as those of the "pithiatique" group (paralyses, contractures, etc.), which one is unable to reproduce by suggestion.

It is pointed out that reserve should be exercised with respect to visceral hæmorrhages and secretory disturbances as being symptoms of hysteria.

M. Cestan quotes a case of anuria present in a hysterical patient, but he adds that he could not affirm its hysterical nature.

Babinski and Dupré state that they have never been able to produce "hysterical fever" by suggestion, and so far do not accept the observations made under this heading.

J. GEORGE PHILLIPS.

FURTHER CONTRIBUTIONS TO OUR KNOWLEDGE OF TORTICOLLIS MENTALIS (HYSTERICUS.) Sectio. (Weitere Beiträge zur Kenntnis des Torticollis mentalis (hystericus), mit einem Sektionsbefund.) JENÖ KOLLARITS, *Zeitschrift. f. Nervenheilk.*, Bd. 35, Hft. 1 und 2, S. 141.

THIS paper gives a summary of the points in favour of the psychical origin of spasmodic torticollis. Three new cases are described, one of which appeared to be a pure traumatic neurosis. In none was there any sign of labyrinthine disease, which Curschman has put forward as a cause of torticollis. There is also the account of a sectio on a case previously reported. The only change found in the nervous system was a very distinct degeneration in the column of Goll, which it is very difficult to connect with the disease in question.

J. H. HARVEY PIRIE.

MIGRAINE, AN OCCUPATION NEUROSIS. GEORGE L. WALTON, (540) *Journ. of Am. Med. Assoc.*, July 18, 1908, p. 200.

DEFINING an occupation neurosis as "a condition resulting from overuse of certain parts," the author considers that migraine is an occupation neurosis, and involves—(1) the visual centres; (2) the centres of accommodation; (3) the intrinsic and extrinsic muscles of the globe; and (4) the muscles outside the orbit which are called into play in the effort required for accurate vision, principally the corrugator supercilii and occipito-frontalis, and also the muscles inserted in the occipital region, which serve to steady the head. Migraine results, in individuals of neurotic inheritance, from overuse, or use under the handicap of refractive error, of the parts concerned in vision. In cases of extreme susceptibility migraine may even appear without error of refraction from moderate use of the eyes. He believes that the vasomotor phenomena are secondary, and that the vomiting "represents a protective effort on the part of nature."

J. A. GUNN.

DOUBLE OPTIC NEURITIS FOLLOWING VARICELLA. (Névrite optique double suite de varicelle.) P. CHAVERNAC, *Ann. d'oculistique*, 1908, p. 52.

A BOY, aged 11 years, had an attack of varicella in February 1905, for which he was kept in bed a few days. As soon as he was convalescent he noticed that his sight was impaired. Within forty days he was unable to read, and by June he could not count fingers at a metre's distance. Optic neuritis was diagnosed by

several eminent ophthalmologists, and he was subjected to intense mercurial treatment from March to November. In November the mercury was omitted. Fifteen subconjunctival injections of hetol were then given at two to three days' interval, and considerable improvement resulted. Chavernac thinks that mercury should be used with great care in optic neuritis of infectious nature. In the present case it was inactive and perhaps harmful, since mercurial intoxication was superadded to the varicella poison.

J. D. ROLLESTON.

THE PHENOMENON OF CHARLES BELL. FUMAROLA, *L'Encephale*, (542) May 1908, p. 385.

THE author examined thirty-two cases of facial paralysis. In thirteen cases the eye rotated upwards and outwards—eight times upwards, six times upwards and inwards; in five cases the eye remained immobile; once it rotated downwards and inwards, and once directly inwards. He considers the phenomenon to be purely normal, rendered visible by the paralysis, and of no prognostic significance whatsoever.

S. A. K. WILSON.

HERPES OF THE MEMBRANA TYMPANI DUE TO ZOSTEROID
(543) **AFFECTION OF THE PETROSAL GANGLION.** T. J. ORBISON, *Journ. of Nerv. and Ment. Dis.*, Aug. 1908, p. 500.

EARACHE, tinnitus and deafness in the left ear. An herpetic vesicle on the posterior surface of the membrana tympani together with similar vesicles in the palatine and alveolar regions.

The author refers to the sixty cases collected by J. Ramsay Hunt, in which peripheral facial paralysis and zoster co-existed and were attributed by that writer to inflammation of the geniculate ganglion. He remarks that the petrosal ganglion of the glossopharyngeal nerve plays the same role as the geniculate on the 7th, and argues on anatomical grounds that it is the former ganglion which is the seat of the lesion in the case he reports.

EDWIN BRAMWELL.

THE SYMPTOMS AND BASIS OF WORD-DEAFNESS. (Ueber (544) *Erscheinungen und Grundlagen der Worttaubheit*.) QUENSEL, *Deutsche Zeitschr. f. Nervenheilk.*, 1908, Bd. 35, S. 25.

THE author describes three cases of word-deafness, two of them with autopsy, and after a long discussion of the phenomena sums up his chief conclusions as follows:—

(1) The cortical area, whose destruction produces word-

deafness, includes the posterior half of the first temporal gyrus, together with the transverse temporal gyrus, on the left side in right-handed individuals. The transverse temporal gyrus must be completely destroyed to produce total word-deafness.

2. Total word-deafness can occur—(a) In cases of subcortical lesion with complete interruption of the auditory radiation and of the corpus callosum in the region of the word-hearing area. Partial interruption of the auditory radiation, with total interruption of the corpus callosum, leaves the hearing and comprehension of words intact, provided the cortex of the transverse temporal and first temporal gyrus be undamaged. (b) Total word-deafness occurs in a lesion entirely or partially cortical, if the left transverse temporal gyrus is isolated or included in a larger lesion. This result will also occur in a right-sided lesion, even though the process of hearing is not completely lost.

3. Cases of partial lesion of the transverse temporal gyrus show partial word-deafness, with the power of repeating correctly words of one or two syllables, but failure with other words.

4. The power of repeating words heard in partial word-deafness occurs only when lesions are also present in Broca's convolution or in the motor area.

5. Intact power of repeating words heard in certain cases of word-deafness shows that besides parts of the transverse gyrus, other parts of the first temporal gyrus have survived. The converse, however (preservation of function when these areas are spared), does not necessarily occur.

6. Disorders of reading are not necessarily associated with destruction of the above area for word-deafness, although loss of comprehension of what is read, with preservation of the power of reading aloud, may be expected.

7. Alexia, complete or partial (except the variety included under paragraph 6), is observed almost exclusively in cases where the lower parietal region, especially the angular gyrus, has been destroyed, at least partially.

8. The loss of power of repeating words heard and of reading are not necessarily proportional to the word-deafness.

9. Anamnestic and paraphasic faults of spontaneous speech are hardly ever absent in word-deafness, though their intensity is variable.

10. The power of spontaneous expression by writing in cases of word-deafness is dependent on (a) impairment of spontaneous speech, and (b) impairment of reading. The loss of power of writing to dictation is dependent on (a) the condition of spontaneous speech, and (b) the degree of word-deafness.

11. When the power of copying is lost in cases of word-deafness,

this corresponds either to a total alexia or to a concomitant motor aphasia or apraxia.

12. Cases of word-deafness with marked apraxic phenomena are associated with implication of the supra-marginal gyrus.

13. The following points are in favour of the existence of a well-defined form of word-deafness :—(a) The occurrence of word-deafness as a clinical syndrome. Hitherto only one case has been shown to be due to a subcortical focus of disease. (b) The so-called transcortical sensory aphasia of Wernicke is a clinical syndrome, mostly transitory, and due to a diffuse morbid process. As a direct stabile focal symptom it is not firmly established.

Against its existence is the fact that so-called cortical sensory aphasia may be of most varied clinical forms, whose phenomena vary according to the variation in the positions of the anatomical lesions.

14. As to the division of cases of word-deafness according to their underlying anatomical lesions, it is best to start from the proposition that we have in the brain cortex a relatively widespread area which has to do with the association of the auditory speech function, into which area the auditory projection-fibres enter. This area—the transverse temporal gyrus—forms a defined centre together with a transition area. Every case of word-deafness caused by a deep peripheral focus may be called “pure perceptive,” a variety which is total, pure, and stabile. On the contrary, every case of word-deafness caused by a central focus is necessarily incomplete, complicated, and generally capable of improvement—the “pure associative” variety. The cases where the transverse gyrus is more or less implicated produce the mixed form of perceptive-associative word-deafness, which may be partial or complete, permanent or curable.

15. The conditions under which word-deafness may improve, though it seldom passes off completely, cannot yet be formulated with certainty. Stabile cases are often associated with bilateral lesions of the word-hearing centres, but sometimes with unilateral destruction of the transverse gyrus as part of a more extensive lesion. Probably the implication of the corpus callosum is of great importance. In cured cases the word-hearing area has never been bilaterally destroyed.

PURVES STEWART.

A CASE OF PARAPHASIA. GRASSET and RIMBAUD, *Rev. Neurol.*, (545) June 30, 1908, p. 577.

IN this case the patient exhibited typical paraphasia, and while he read and wrote with difficulty, there was neither agraphia nor word-blindness. There was not the slightest indication of word-

deafness. Nevertheless at the autopsy a softening of the first left temporal convolution was found, towards its posterior end. This lesion is perhaps rather further back than the middle part of the convolution, destruction of which is supposed to produce word-deafness. Minute examination of the brain revealed arterial disease and microscopical cortical changes in various areas, which to the naked eye appeared quite normal. The importance of this cannot be overlooked in any discussion of the relation of the clinical symptoms to the pathological facts.

S. A. K. WILSON.

RELATIVE EUPRAXIA IN RIGHT HEMIPLEGIA. (*Relative (546) Eupraxie bei Rechtsgelähmten.*) MEYER, *Deutsche Med. Woch.*, June 25, p. 1143.

THE first patient was a man with slight right hemiplegia, probably of syphilitic origin. In proportion to the degree of paralysis of the right arm, his capacity for performing various movements with it was astonishingly good. While he could not lift his arm, on request, much higher than his shoulder, he was able to lift it higher, sometimes by as much as 10 centimetres, if an object was held up for him to grasp. His caligraphy with the right hand was quite good.

On the other hand, a second patient showed exactly the reverse conditions. With considerable conservation of strength in the paresed right arm he was unable to perform many movements, nor could he reach as high for an object as without one. The first case was probably due to a lesion of the basilar artery, whereas other signs in the second case pointed to a lesion of the cortex. The author sums up with the loose statement that the further away the lesion is from the cortex, the less the degree of apraxia.

S. A. K. WILSON.

THE ABDOMINAL REFLEX. (*Contributo allo studio del riflesso (547) addominale.*) A. BALDI, *Il Policlinico*, Aug. 2, 1908, Fasc. 31, p. 965.

BALDI investigated the abdominal reflex in the various conditions in which the physiological integrity of the skin had been impaired, viz., by pregnancy, new growths, laparotomy, or such diseases as tuberculous peritonitis or small-pox. In twenty-seven primiparæ in whom delivery had taken place not more than six months previously the reflex was absent. In forty-one primiparæ who had been delivered from two to five years previously the reflex

was easily obtained. Multiparae, on the other hand, were found to have lost their reflex after the birth of their third child. The reflex was also absent in all the cases in which the sensory conductivity of the skin had been interfered with by tumours, operation wounds, tuberculous peritonitis or small-pox.

J. D. ROLLESTON.

HEREDITARY TREMOR. (Tremblement héréditaire, rappelant (548) celui de la sclérose en plaques.) DROMARD, *L'Encéphale*, July 1908, p. 45.

THE patient is an old man of 73, who has been an inmate of the asylum of Clermont since 1862. Tremor of the arms and head, resembling the intention tremor of disseminated sclerosis, was noted in his case at least as early as 1887. At the present time his mental powers, in spite of his age, do not appear enfeebled. He presents a highly advanced intention tremor of the arms, of wide amplitude, increasing with the progression of the movement, a certain spasmodicity of the lower extremities, staccato speech, and nystagmus. The superficial resemblance to disseminated sclerosis which the case shows cannot obscure the significance of various points which make that diagnosis impossible.

The real import of the case will be grasped at once when we learn that the patient's father and mother both suffered from an identical tremor, and died at an advanced age. The patient is the second of three children, of whom the youngest was similarly affected.

The author is able to supply illustrations of the handwriting of father, mother, and patient.

S. A. K. WILSON.

RECKLINGHAUSEN'S DISEASE. RAYMOND and ALQUIER, (549) *L'Encéphale*, July 1908, p. 6.

IN all probability Recklinghausen's disease is a congenital affection, either from malformation or defective development, whose clinical evolution is in many ways comparable to that of dermoid cysts. It may remain latent for a variable time, or may progress slowly, or may develop with great rapidity—facts which render an accurate prognosis difficult.

Only rarely are the tumours true neuromata; they are commonly akin to the "false neuromata" of Virchow, that is to say, they are formed of connective tissue. Their structure is fibromatous, fibro-lipomatous, or sarcomatous. The fact of their structure being sarcomatous does not necessarily indicate a rapid development. The sites of the tumours are likewise variable; we

meet with neuro-fibromata, also with perivascular fibromata, and also periglandular fibromata.

The authors have met with fibromata of the skin, in which the vessels and nerves running through the fibrous tissue are perfectly normal; such cases may justifiably be described as "pigmentary dermato-fibrosis."

S. A. K. WILSON.

COMPLETE RADICULAR PARALYSIS OF THE BRACHIAL (550) PLEXUS WITH OCULO-PUPILLARY PHENOMENA, FOLLOWED BY AUTOPSY. (*Paralysie radiculaire totale du plexus brachial avec phénomènes oculo-pupillaires autopsiée trente-six jours après l'accident.*) Madame DÉJÉRINE-KLUMPKE, *Rev. Neurol.*, July 15, 1908, p. 637.

THE patient was a young man who in a bicycle accident sustained a complete paralysis of his right arm. When he was seen, it was cold, cyanotic, and œdematous, with absence of the radial pulse. There was complete loss to all forms of sensation as far as the shoulder, with the exception of a strip running from the axilla down the inside of the upper arm almost as far as the elbow. All the right arm deep reflexes were lost. There was a cervical sympathetic lesion as well, on the same side. The patient died during an operation undertaken to relieve the symptoms, and the parts were subsequently dissected with care. The axillary and sub-clavicular arteries were found to be thrombosed. Behind the first rib the trunk common to the fifth and sixth cervical roots was found to be elongated; the seventh and eighth cervical and the first dorsal roots were completely torn across; the ganglia corresponding were wrenched out of their bed. Vessels and nerves alike were embedded in a mass of fibrous tissue (the operation was thirty-six days after the injury). The only part of the sympathetic involved was the communicating branches of the roots just mentioned.

It is unusual to have the lower roots destroyed by accident while the upper are more or less intact. Oculo-pupillary phenomena are encountered only when the ramus communicans of the first dorsal is involved.

S. A. K. WILSON.

XVIII. CONGRÈS DES MÉDECINS ALIÉNISTES ET NEUROLOGISTES DE FRANCE. DIJON, August 3-8, 1908, *Rev. Neurologique*, August 30, 1908.

AMONG many interesting communications the following are worthy of notice:—

Léri has noted aplasia of the suprarenals as a constant accom-

paniment of anencephaly. Anencephaly is not an arrest of development, but the consequence of an inflammatory disease of the central nervous system. The suprarenal aplasia is elective, since the other organs are intact. It is conceivable that the further production of lecithin by the gland becomes unnecessary when the brain is destroyed, and that therefore the gland undergoes atrophy.

In seventeen cases of epilepsy, Claude and Schmiergeld found alterations in the thyroid gland in all; in twelve the normal glandular structure was entirely changed, there being areas of atrophic sclerosis, with zones of compensatory hypertrophy.

Cases of psychasthenia and other mental troubles (phobias, anguish, etc.) associated with glandular troubles ("instabilité thyroïdienne," "hyperthyroïdie," "hypo-ovarie," etc.), and treated, in some instances, with considerable success by the appropriate glandular extract, are reported by Sollier and Chartier, and Lévi and Rothschild. Some of the cases are very convincing.

Parhon and Urechia observed catatonic phenomena in dogs deprived of the "thyro-parathyroid apparatus." Muratow had already found changes in the thyroid gland in catatoniacs, but Kraepelin failed to register improvement by thyroid treatment.

In the treatment of writers' cramp Meige recommends that the patient's caligraphy should be "peu, lent, rond, gros, droit," i.e. he should write little and slowly, and his letters should be round, large, and upright. In the following out these simple instructions Meige has noted improvement in various cases.

S. A. K. WILSON.

PSYCHIATRY.

JUVENILE GENERAL PARALYSIS. (*Paralysie générale juvénile* (552) *chez un sujet de 23 ans.*) JOFFROY, *L'Encéphale*, July 1908, p. 1.

THE patient was a case of physical infantilism, who, in spite of his twenty-three years, had the appearance of a boy of thirteen or fourteen. While under observation his memory began to fail; his hands, tongue, and facial muscles became tremulous, his speech slurring and almost unintelligible; his pupils became unequal and reacted sluggishly to light; a marked lymphocytosis was found in the cerebro-spinal fluid. There was no history of preceding syphilis.

The author remarks on the frequency of the pre-existence of physical infantilism in cases of juvenile general paralysis.

S. A. K. WILSON.

ALCOHOL AND MENTAL DISEASE W. R. DAWSON, *Dub. Journ.*
(553) of *Med. Sci.*, June 1908.

APART from personal bias, the uncertainty existing with reference to the effects of alcohol is largely due to insufficient appreciation of the influence of individual diathesis and of the varying effects of different doses. On the whole, it appeared that for normal persons, and under appropriate circumstances, small doses properly diluted and taken with food not too frequently were physiologically beneficial, but for neuropaths, and with larger or more frequent doses, the case was otherwise. Alcohol taken occasionally in large doses—the “convivial drinking” of Sullivan—produced less serious effects than in moderate doses very frequently repeated, though in highly neuropathic persons serious results might arise. The latter mode of drinking, the “industrial drinking” of Sullivan, was due to the influence of alcohol in facilitating for a time coarse muscular exertion. It produced in the long run progressive mental and bodily degradation, between which and technical insanity no hard and fast line could be drawn. Moreover, of the technically insane all did not enter asylums, and therefore a complete estimate of the amount of mental disease due to alcohol was at present impossible. Even as regarded the insane in asylums, much difficulty existed in arriving at a correct conclusion, and for a variety of reasons it was almost certain that the amount of mental disease really chargeable to alcohol had been over-estimated. About 15 to 20 per cent. of cases seemed to be the proportion usually so attributed, but probably 10 per cent. would be nearer the mark. (In Ireland the average percentage of cases so caused in the previous five years had been stated at 9·90.) The regional distribution of insanity did not correspond with that of either alcoholism or drunkenness. On the other hand, the cases in which alcohol acted as a contributory cause must be numerous, owing to the susceptibility to its action of persons with hereditary or acquired brain weakness. The mental *symptoms* most characteristic of alcoholic insanity were confusion with hallucination in the acute cases, a tendency to impulsive action, suspicion passing into delusion, and loss of recent memory with a tendency to supply its place by fabrications. The most purely alcoholic *forms* of insanity were delirium tremens, Korsakoff's psychosis, hallucinatory-confusional insanity, and some cases of alcoholic dementia. The other alcoholic psychoses presupposed a greater or less degree of neuropathic tendency, beginning at the top with *mania a potu*, trance states, and dipsomania, and passing down through alcoholic paranoia, mania and melancholia, to dementia. As regarded treatment, the essential was total abstinence, which must be enforced for a time—

a minimum of two years had been found necessary at Ennis Reformatory. It was therefore highly desirable that such cases on recovery of their sanity should be transferable from the asylums to inebriate reformatories, and legally detainable therein for a time. For all neuropaths total abstinence afforded the only hope of continued well-being.

AUTHOR'S ABSTRACT.

THE SENSE OF TIME IN KORSAKOFF'S PSYCHOSIS. (Zur (554) **Kenntniss des Zeitsinnes bei der Korsakoffschen Geistesstörung.**) ADALBERT GREGOR, *Monatsschr. f. Psychiat. u. Neurol.*, June 1908.

IN this paper the author describes a series of experiments he conducted in a case of Korsakoff's psychosis. The patient presented marked disturbance of time consciousness, and the object of the investigation was to obtain an exact record of the sense of time he possessed. As controls to the experiments, one normal and two pathological individuals, who did not present the particular disturbances in question, were subjected to the same tests.

The results are given in tabular form.

A. HILL BUCHAN.

DISTURBANCES OF THE PSYCHIC FUNCTIONS IN CASES OF (555) UNILATERAL NASAL OBSTRUCTION. (Ueber Störungen der psychischen Funktionen bei einsitiger Behinderung der Nasenathmung.) WILH. ANTON, Prague, *Prag. Med. Wchnschr.*, June 1908.

THE connection between disturbances of mentality and nasal obstruction was recognised as early as 1868, and described under various titles—"mental work made difficult," "psychic depression," etc., while in 1888 Guye gave a definite picture of the condition, the principal symptom of which was incapacity to direct one's attention to a given object, and bestowed upon it the name "Aprosexia." Mental impressions are made with difficulty and are easily lost; there is a continuous or intermittent feeling of pressure in the head; giddiness and signs of increased nasal reflex excitability such as fits of sneezing or "trigeminal" cough may be present. Guye's cases referred to adenoid post-nasal growths in children; but other causes of the condition are found in nasal catarrh, crests or deviations of the nasal septum, and turbinal hypertrophy. After removal of the cause of nasal obstruc-

tion the symptoms rapidly disappear. Kafeman in his psychological experiments used a nasal obturator, and found that mathematics were made specially difficult; the perception of visible things was only slightly influenced, but the memory of these objects was not nearly so good as in normal conditions: in all the above cases both nasal cavities were obstructed. Anton now records two cases of unilateral nasal obstruction in which there was disturbance of psychic function; removal of the obstruction resulted in cure in both instances. The first case was that of a schoolboy, aged 17, who suffered from nasal (choanal) polypi on the left side: his mathematical studies were specially difficult, and, as he could not understand his lessons, he often went to sleep over his books. From the report of the nasal examination it would seem that there must have been some obstruction of both sides, as it is stated that the nasal cavities were narrow, and that nasal catarrh was present along with enlargement of the posterior ends of the turbinal bodies. After removal of the polypi the nasal symptoms disappeared at once and the mental condition slowly improved. In the second case—that of a girl of 16 years—there was congenital bony occlusion of the left choana: the patient suffered from left-sided headache and had been a somnambulist since childhood: the right side of the nose was normal in this case. After operation the sense of smell at once returned in the left nostril, and some days later the sleep became quiet.

Guye has called attention to the connection between the subdural lymph space and the lymphatics of the nasal mucosa, which has been proved by Axel, Key and Retzius. The theory is that in cases of nasal obstruction there is not a sufficient loss of fluid from the surface of the nasal mucous membrane: the cause of the nasal obstruction (polypi, etc.) may also mechanically interfere with the lymph-flow. In these ways the elimination of effete products is to some extent prevented and "retention-exhaustion" of the cerebral cortex occurs.

Zarniko, on the other hand, considers that, in comparison with the other methods of drainage, the lymph flow from the brain to the nasal mucous membrane is of small importance, that "aproxexia" occurs in only a small proportion of cases of nasal obstruction, and finally that it is absent in conditions where the lymphatic streams in the nose are greatly reduced, *e.g.* atrophy of the nasal mucous membrane. For these reasons Zarniko thinks that aproxexia is only a special form of neurasthenia which is produced by nasal obstruction and its consequent disturbance of sleep. Anton is of opinion that his two cases support Zarniko's theory, and points out that his first patient was worried and slept badly. The whole discussion can be summed up in two questions:—(1) Does nasal

obstruction directly cause psychic disturbance by interfering with the lymph flow from the brain; or (2) does nasal obstruction act by creating or increasing a condition of neurasthenia of which "aprosexia" only forms a part?

J. S. FRASER.

THE PROTECTION OF SOCIETY FROM CRIMINAL LUNATICS.

(556) (*La difesa sociale dagli alienati criminali.*) TAMBURINI, *Riv. di Sper. Freniatr.*, Vol. xxxiv., Fasc. 1-2, p. 274.

AN article dealing with questions of Italian law, in which the author advocates the English system of dealing with criminal lunatics, especially in regard to the question of their discharge.

ERNEST JONES.

THE ABOLITION OF THE USE OF TOBACCO AMONGST THE

(557) **INSANE AT THE LUCCA ASYLUM.** (*L'Abolizione dell'uso del tabacco per gli alienati nel manicomio di Lucca.*) ANDREA CRISTIANI, *Riv. di Sper. di Freniatr.*, Vol. xxxiv., Fasc. 1-2, p. 286.

ASSURED by the success of his experience in abolishing the use of alcohol in his asylum, Cristiani has done the same for the past year or so with tobacco. The results, he claims, are good, and there are no disadvantages. With each individual the quantity is first gradually reduced before complete cessation is enforced.

ERNEST JONES.

TREATMENT.

THE CLINICAL USE OF HYPNOIDIZATION IN THE TREAT-

(558) **MENT OF SOME FUNCTIONAL PSYCHOSES.** J. E. DONLEY, *Journ. of Abnorm. Psy.*, Aug-Sept. 1908, p. 148.

THIS paper contains an account of the treatment of certain psychic disorders by the hypnoidization method of Dr Boris Sidis. The desired state was induced in the patients by placing them on a couch, the head of which was close to a faradic wall plate. They were then requested to listen to the monotonous vibration of the ribbon rheotome, and to concentrate their attention either upon nothing or upon the ideas suggested by the physician. In a few minutes they sank into the hypnoidal state, during which it was possible to obtain information valuable for diagnosis and to give therapeutic suggestions.

Descriptions are given of five cases in which the method was used with marked success.

MARGARET DRUMMOND.

NERVE ANASTOMOSIS IN INFANTILE PARALYSIS. KARL
(559) OSTERHAUS, *Med. Rec.*, July 11, 1908.

THIS paper consists of a review of the present methods in use for the treatment of infantile paralysis and the report of one case in which nerve anastomosis was performed. The case was one of slight right talipes equino varus of four years' standing in a boy aged ten years. At the operation a bundle of nerve-fibres was isolated from the internal popliteal nerve and a similar bundle from the external popliteal nerve, the former being centrally, the latter peripherally, attached; the two nerve bundles were then united end to end with fine catgut. At the same time the Tibialis anticus, the tendo Achilles, and the plantar fascia were divided subcutaneously. The limb was enclosed in plaster for six weeks.

At the end of four months there was a very slight improvement in the function of the limb.

Systematic massage, electricity, and baking were carried out in the after-treatment.

D. P. D. WILKIE.

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Review of Neurology and Psychiatry

Original Articles

LEPTOTHRIX INFECTIONS. A CASE OF PYÆMIA WITH MENINGITIS, AND NOTES OF TWO SIMILAR CASES.

E. SCOTT CARMICHAEL, M.B., F.R.C.S.E.,
Assistant Surgeon, Royal Hospital for Sick Children.

With Pathological Report by
Drs JAMES RITCHIE and STUART MACDONALD.

THE following case came under my notice two years ago. In April 1906, in consultation with Dr Thyne, I saw an infant, æt. 4 months, who had suffered from gastro-intestinal symptoms for a fortnight. Two days previously the child began to suffer from abdominal pain, which suggested to Dr Thyne the possibility of peritonitis. The abdomen was swollen, tympanitic, and rigid, the child presenting locally all the symptoms of general peritonitis. The general condition of the child, however, was not such as one would associate with a general peritonitis due to the more common pyogenic organisms.

The abdomen was opened, and general peritonitis, evidently of some duration, was present. The pelvis was full of a somewhat serous pus, and there were dense adhesions between the coils of intestine. These were especially so around the cæcal region, so that no attempt was made to find the appendix. Multiple drainage was used for pelvis and lumbar regions.

The child made an uninterrupted recovery, and was dismissed weeks afterwards completely cured. An ischio-rectal abscess developed a month later, but since then the child has been in good health.

The pus from the abdominal cavity was submitted to bacteriological examination. Mr Richard Muir reported that the infection was a mixed one, but that the predominant organism was a leptothrix, probably of intestinal origin.

The interest of this case lay in the fact that the child was very young (four months), and recovered after an attack of generalised acute peritonitis. The child was never so seriously ill as might be expected in a case of generalised peritonitis.

It has since been in good health.

Recently it has been my good fortune to meet with a similar case. I am indebted to Dr James Ritchie and Dr Stuart Macdonald for the pathological-bacteriological investigation of the case.

A child, æt. 18 months, was sent by Dr Dickie to Dr John Thomson's ward on May 6, 1908. It had been ailing for about ten days with gastro-intestinal symptoms. Two days before admission, swelling appeared in the right lower limb. Dr Thomson asked me to see the case with him.

There was general œdema of the limb up to the lower third of the thigh downwards. There was no redness or inflammation except for a small area about the size of a shilling in front of the external malleolus. The temperature was 104° F. The diagnosis lay between an acute osteo-periostitis of the tibia or fibula and a subcutaneous œdema of septic origin.

The child, though ill, was not so bad as to lead one to suppose that there was disease of the bone, and the absence of redness and the general appearance of the limb was not characteristic of acute osteo-myelitis. There was no localisation of the swelling. The knee and ankle joints were freely movable without pain, although they participated in the œdema.

Although the clinical symptoms were against osteo-myelitis, one did not feel justified in not exploring the bones of the leg.

Incisions were made down to the bone over the tibia and fibula, but except for a general œdema of the subcutaneous tissues, there was no evidence of more deep-seated inflammation. The reddened area was incised and a small pocket of pus was

found under the common extensor tendons, in front of, and independent of, the ankle joint.

This was examined bacteriologically by Dr James Ritchie.

The child's condition continued much the same for the next three days, the temperature remaining persistently about 103° F. The general condition, however, was well maintained. Bier's treatment with the Saug-Apparat was carried out during this period.

Discharge from the right middle ear, with bilateral swelling of both elbows, was then noticed. The joints were markedly distended, but there was no redness or signs of inflammation or marked local rise of temperature.

Both joints were drained, and contained a large quantity of thin, odourless, and rather pale creamy-coloured pus.

These were again examined bacteriologically by Dr James Ritchie.

The child's condition became distinctly worse. There was very little discharge from the joints, much less than is present in streptococcal or pneumococcal affections. The child lived for a week longer with continued high temperature. Two days before death there was some swelling of the cervical glands.

Lumbar puncture was performed and a turbid fluid drawn off, which was examined bacteriologically.

A point of interest in these cases was that the general condition of the child in each case was not gravely affected while the leptothrix infection was localised. In the case of the first child, though young, the recovery was complete. In the second child, though there was high temperature, the general condition remained fairly good until there was general systemic infection, as shown by the otitis media and the double elbow-joint affection.

The corresponding infection by staphylococci gives rise as a rule to much more systemic derangement.

The œdema of the leg was not associated with any redness; there was no evident active inflammatory change; the condition of the limb resembled much more that of an œdema of cardiac or renal origin than due to an inflammatory condition. I have seen from time to time in the out-patient department children brought up with a similar œdema of the hand and forearm, unassociated with inflammatory change. In one case I made an

incision and endeavoured to cultivate an organism, but with negative result.

On looking back on these cases I cannot but think that they may be of similar origin. The œdema often persists for a week or two without affecting the general condition of the patient to any marked degree, and then gradually subsides.

We have considered these often of a mild streptococcal nature, without being able to grow such an organism.

The want of local inflammatory reaction was markedly seen in the case of the elbow-joint swellings. It is unlikely that these had been overlooked for more than a day or two, but the appearance much more resembled that of a tuberculous affection. It was not like an acute arthritis due to the more common pyogenic organisms.

The pus was thin, odourless, and of a pale creamy colour.

In the abdominal case it was thin and more serous, odourless, much more like the fluid of a pneumococcal condition.

The organism, if it is the cause of these conditions, does not appear to be an acutely virulent one, but can lead to a general pyæmia, as shown in the last case.

These cases, however, seem to show that the organism is generally associated with other organisms, such as the pneumococcus.

It is also of interest to note that both cases were ushered in by gastro-intestinal symptoms.

CASES 3 AND 4.—Drs John Thomson and Fowler have kindly permitted me to refer to two cases under their charge, the bacteriological findings in each case being similar to those above recorded.

Without referring at any length to these cases, I may say that they presented the symptoms and course of a post-basic meningitis.

In Dr Fowler's case head retraction appeared one week after the onset of the illness, which was ushered in with vomiting. Lumbar puncture on six occasions was performed, and indefinite diplococci found. The child was treated with Flexner's serum. It died five weeks after admission to hospital.

The bacteriological examination in these cases, carried out

by Dr Stuart Macdonald, gave a similar finding to that described fully in Case No. 2.

Post-mortem and Bacteriological Report. CASE 2. By Dr James Ritchie and Dr Stuart Macdonald.

There was a purulent sub-dural meningitis over left hemisphere, a purulent pia-arachnoiditis in patches over the surface of the brain, and spinal meningitis about the lumbar cord.

From the abscess in front of the ankle joint, the elbow joints, cerebro-spinal fluid and peripheral blood, a gram negative bacillus was obtained in long filamentous forms.

It was mobile, grew on all media, did not liquefy gelatine, and somewhat resembled the typhoid bacillus in its characters, giving a slight acid reaction in milk and glucose. There was in the case evidence of a terminal pneumococcal infection. The bacillus was pathogenic to monkeys and mice, very slightly to guinea-pigs, and apparently to rabbits. It appears to break down the resistance of the animal to other pathogenic organisms. The importance of the case seems to rest in the production of the pyæmic process by a probably hitherto undescribed organism.

THE EXAMINATION OF CEREBRO-SPINAL FLUID IN GENERAL PARALYSIS FOR PURPOSES OF DIAGNOSIS.

By HAMILTON C. MARR, M.D., F.F.P.S.G.,
Medical Superintendent of the Glasgow District Mental Hospital, Lenzie.

"A SLIGHT tremor of the lips, and a hesitating utterance, as if the lips and tongue had no grip (so to speak) over the consonants, will, along with a peculiarity in the gait, an unusual stillness in the muscles of expression, and a slight disparity of the pupils, reveal with almost absolute certainty an early stage of one of the most hopeless of diseases—general paralysis of the insane."¹

The diagnosis of general paralysis is not, however, so easy. The above signs are found associated with forms of chronic

¹ Gairdner, "The Physiognomy of Disease," from *Finlayson's Clinical Manual*.

alcoholism, neurasthenia, advanced cerebral arterio-sclerosis, cerebral apoplexia and softenings, syphilitic affections, tumour of the brain, and lead poisoning. Many of these mental conditions are recoverable, but as yet no hope of improvement can be held out to the general paralytic. Under these circumstances it is important to be able to differentiate between the several conditions named. An examination of the cerebro-spinal fluid is of great consequence in reaching a diagnosis. In an investigation extending over three years, and embracing an examination of the cerebro-spinal fluid of all varieties of insanity, fifty-three cases of general paralysis have come under my observation. Elaborate physical and chemical examinations have been made in every case, and in this paper I will draw attention more particularly to a summary of the cytology, the significance of the presence of the serum-albumen, and the bacteriological conditions of these cases.

In fifty-two of the cases a lymphocytosis was noted. The exception was a case of juvenile general paralysis, where the cerebro-spinal fluid exhibited a few lymphocytes, but not a sufficient number to constitute a lymphocytosis.

The methods of discovering the presence of a lymphocytosis are varied. That suggested by Widal¹ is best for clinical purposes. He applies the term "lymphocytosis" when the cerebro-spinal fluid contains six to ten lymphocytes in each field of the microscope when an oil immersion lens is used. Ten several fields should be examined before a decision is reached. An objection has been raised that the cellular elements do not stain well by Widal's method, but this objection is averted by the use of Leishman's stain, which allows of a differentiation between the cellular elements. These elements are generally large and small lymphocytes, and in each field one or two plasma cells are found—that is to say, large cells with a nucleus stained blue like that of the lymphocyte, the stained material radiating from the nucleolus toward the periphery of the nucleus. The protoplasm of the cell stains a reddish pink.

The only form of insanity of those enumerated in which a lymphocytosis occurs is syphilitic insanity, and there only when the syphilitic condition is active. In three cases of syphilitic insanity observed, two showed the presence of a few lymphocytes in the cerebro-spinal fluid ; in the third there was a lymphocytosis.

¹ Widal, *Revue Neurologique*, No. 6, March 30, 1903.

The first syphilitic patient died, and a post-mortem examination revealed a gummatous deposit in the left frontal region. The second also died, and a large mass of tumour occupied the right motor cortex, causing crossed hemiplegia. The third case, in which lymphocytosis was noticed, made a good recovery. The lymphocytosis and serum-albumen, which were present in the cerebro-spinal fluid, disappeared as a result of anti-syphilitic treatment. The first of these three cases was the only one which could have been mistaken clinically for a case of general paralysis.

The amount of serum-albumen present in the cerebro-spinal fluid of general paralytics varies from .05 per cent. to .4 per cent.; the average amount is .15. The presence of serum-albumen is common to many insanities. I have invariably found it associated with chronic alcoholism, and have discovered it in some cases of epilepsy and in many of organic brain disease, whether due to cerebral softening, hæmorrhage, or senile decay.

The presence of serum-albumen is apparently associated with an inflammatory or congested condition of the cerebral meninges. Its persistence is an indication that the patient is not likely to recover by his mental affection. Thus, in one case of lead poisoning and in five cases of alcoholism, all of which ultimately recovered, serum-albumen varying from .1 to .25 per cent. was present at the onset of illness, but this disappeared gradually in every case until there was only a trace of albumen in one of the alcoholic cases.

A bacteriological examination has been made in every case. At first culture tubes of blood agar were used, and into these a few drops of cerebro-spinal fluid were passed from the needle at the time when the fluid was being collected from the spinal canal. Latterly, in view of Dr Ford Robertson's use of byno-hæmoglobin, byno-hæmoglobin agar and blood agar have been used. Dr Ford Robertson states that he has obtained the *bacillus paralyticus* in the cerebro-spinal fluid of four general paralytics. In this respect my observations are different. I have not obtained a bacillus or other organism from the cerebro-spinal fluid of any of the fifty-three cases under review. In eighteen cases tubes of blood agar and byno-hæmoglobin agar were used as culture media; in the remainder, blood agar tubes only. The cerebro-spinal

fluid, after being received into a sterile tapered tube, had the deposit carefully examined for organisms, with negative results.

So far as insanity is concerned, the presence of lymphocytes to the extent of causing a lymphocytosis, and plasma cells, is diagnostic of general paralysis. The simplicity of lumbar puncture, and the absence of ill effects in withdrawing 10 c.cm. of cerebro-spinal fluid, which is sufficient to form a diagnosis, make it an important means of investigation in many mental cases which might, from their clinical symptoms, be classed as incurable.

Abstracts

PHYSIOLOGY.

AN INVESTIGATION OF THE PRINCIPLES UNDERLYING
(560) **WEIGERT'S METHOD OF STAINING MEDULLATED**
NERVE. J. LORRAIN SMITH and W. MAIR, *Journ. Pathol. and*
Bacteriol., Vol. xiii., 1908, p. 14.

CHOLESTEROL FLUID CRYSTALS, AND MYELIN FORMS.
(561) CHARLES POWELL WHITE. Dreschfeld Memorial Vol. *Publica-*
tions Univ. of Manchester, Med. Series, No. 8 (*Med. Chron.*,
March 1908).

LORRAIN SMITH and Mair have shown that Weigert's method of staining medullated nerve can be applied to fat. At a meeting of the Pathological Society of Great Britain and Ireland in June 1907, they showed sections of adipose tissue, fatty liver, fatty heart, kidney, etc., in which the fat globules had been stained and differentiated with the same degree of clearness and definition as is obtained in the case of medullated fibres in sections of spinal cord. The sections were cut by the freezing microtome after formalin fixation, and were then exposed to the action of strong solutions of bichromate. Similar results were obtained with cigarette papers which had been smeared with pure olein and oleic acid. The bichromate solution action acts much more rapidly on myelin substance than on ordinary fats. In both cases chromium compounds are formed which are insoluble in alcohol, ether, etc., and which at the same time are capable of forming coloured lakes with hæmatoxylin. These results suggested that Wlassak's

view that protagon is the substance in the nerve sheath on which the staining by Weigert's method depends required reconsideration.

An extended series of observations on the action of bichromate solutions on various fats, fatty acids, alcohols, and aldehydes, was therefore undertaken. It was found that in the case of fats and fatty acids, only those which contained an unsaturated grouping of carbon atoms are capable of forming chromium compounds which are able to lake hæmatoxylin; and further, that if the action of the bichromate solution be sufficiently prolonged, these unsaturated fats can be completely oxidised to saturated compounds which are no longer capable of laking hæmatoxylin. It is, therefore, only during a certain stage of the process of oxidation that chromium compounds capable of laking hæmatoxylin are formed. As has been shown by Dr Thorpe, the chromium compounds in question are comparatively loose addition products of chromium trioxide, and are not organic salts of chromium. The difference in the rapidity of the action of bichromate solutions on fats and on myelin substance is due, in part at least, to the cholesterin contained in the latter.

Powell White has shown that mixtures of cholesterin and the higher fatty acids give rise to fluid crystals and anisotropic globules, and on the addition of water produce myelin figures. These mixtures are, in a sense, compounds, the acid probably playing the part of an "acid of crystallisation," but they are not true esters of cholesterin. The anisotropic globules and myelin figures are best developed when the cholesterin and fatty acids are mixed together in equimolecular proportions. It appears probable that it is in the form of this loose combination with fatty acids and other substances that cholesterin most commonly occurs in the body, and that the myelin of the white nerve sheath is in a fluid crystalline condition in virtue of the presence of cholesterin in such combination.

Pure cholesterin is not acted on by bichromate solutions at ordinary temperatures, but a mixture of cholesterin with any of the higher fatty acids which gives myelin forms in water is readily acted on by the bichromate solution. Chromium compounds are formed which are insoluble in alcohol and which lake hæmatoxylin, and the mixture behaves in all respects towards these reagents exactly like the myelin of the white nerve sheath. As in the case of fats, if the action of the bichromate solution be prolonged, oxidation is carried too far for staining purposes, and the saturated compounds formed become incapable of laking hæmatoxylin. These facts lead us to recognise different stages of oxidation in the process of bichromating tissues. The staining with hæmatoxylin after bichromating depends on the presence in

the tissues of various unsaturated substances. At any particular stage of oxidation only certain of these substances are capable of combining with hæmatoxylin, and thus at different stages of the process of bichromating different elements will retain the stain. In the spinal cord the ordinary Weigert effect is only obtained at a particular stage of the process of oxidation by the bichromate solution. If frozen sections of spinal cord from tissue fixed either in formalin or in bichromate be exposed in the incubator to the action of a solution of bichromate saturated at a temperature of 37° C., and examined at intervals of a few days by staining in hæmatoxylin, and differentiating as in Weigert's method or any of its modifications, a remarkable series of changes in the elements which retain the stain may be observed. The first element to stain after bichromating has begun is the medullary sheath, and for a time it alone is coloured. Following this we find the colour reaches the lipochromes in the nerve cells, then the nucleoli, and by the time the myelin sheath has ceased to stain, the axis cylinder stands out clearly stained, and surrounded by a yellow ring of oxidised and bleached myelin. Then the cell body and its processes stain diffusely, and finally the neuroglia takes on a clear blue stain. In other words, by continuing the bichromating process, we can exactly reverse Weigert's method and stain all the elements in the tissue which in the ordinary process are left unstained.

AUTHORS' ABSTRACT.

THE ACTION OF STRYCHNINE AND CAFFEINE. (Ein Beitrag (562) zur Kenntnis der Strychnin- und Koffeinwirkung.) TORATO SANO, *Arch. f. die gesam. Physiol.*, Sept. 10, 1908, p. 381.

It has long been known that a frog poisoned with strychnine reacts to chemical stimulation slightly, if at all, at a time when it shows a hyper-excitability to mechanical or electrical stimulation.

Baglioni has explained this phenomenon as due to the fact that chemical stimulation is less sudden and represents a large number of weaker ineffectual stimuli which must be summated in the co-ordination mechanism. In the strychnine-poisoned frog this power of summation is almost completely lost.

The author suggests, as a result of further experiments, another explanation—namely, that there is, at a certain stage of strychnine poisoning, a diminution in the excitability of the central mechanism for pain sensation, but an increase in the excitability of the central mechanism for touch sensation. Strychnine—and caffeine behaves similarly—resembles a group of poisons, *e.g.* morphine, in increasing the reflex excitability, while at the same time exercising an anæsthetic action.

J. A. GUNN.

THE ANTAGONISTIC ACTION OF PARTICULAR PARTS OF THE
(563) **BRAIN TO STRYCHNINE.** (Ueber das Entgiftende Vermögen einzelner Gehirnabschnitte gegenüber dem Strychnin.)
TORATO SANO, *Arch. f. die gesam. Physiol.*, Sept. 10, 1908, p. 369.

A CERTAIN dose of strychnine was allowed to stand in contact, for fifteen to twenty-four hours, with an emulsion of grey matter of human brain, and the mixture was then injected into the dorsal lymph sac of a frog. Parallel experiments were carried out with the grey matter of motor and of sensory regions. From a large number of such experiments the author draws the following conclusions:—

The cortical grey matter can antagonise strychnine, the grey matter of the motor regions being more effective in this respect than that of the sensory regions, and this property is due to the cell elements. The antagonism is of a chemical nature.

The morphologically and functionally characteristic cell elements of the grey matter of the human cortex probably possess one or more substances differing chemically from one another.

The motor elements of the central nervous system possess a higher affinity for strychnine than do the sensory.

J. A. GUNN.

THE PHYSIOLOGY OF THE PITUITARY BODY. (Sur la physiologie de l'hypophyse.) SALVIOLI and CARRARO, *Arch. ital. de Biol.*, Tome lxix., 1908, p. 1.

THE authors confirm the observations of Schäfer and others that injection of extract of the pituitary body exercises an important influence upon the blood-pressure.

The effects observed are a slight fall succeeded by a rise of blood-pressure. These are to be attributed to the posterior or nervous lobe of the gland, whose extract remains active, although it has been deprived of the epithelial layer associated with it. The action is not one which is common to cerebral tissue generally. Injections of cerebral extracts give negative results.

An influence further is exerted upon the cardiac rhythm resulting in increase of systolic force and slowing of the pulse. When the vagi are cut the blood-pressure curve presents appearances which have been described by Cyon. Periods of slowing and strengthening of the systole alternate with periods during which we have acceleration and diminution of the pulse. There are also to be observed after injection oscillations in the curves forty to fifty seconds in duration, which are due to variations

in vascular tone and are independent of the above-mentioned alternations.

The amount of the injection is an important factor as affecting blood-pressure and cardiac rhythm. With small doses the action is chiefly upon the former, while the latter is principally affected when large injections are made.

When the injections are repeated, the second and later ones have less effect than the first both on blood-pressure and systole.

The extract acts directly upon the vessel walls and not to any considerable extent through the vaso-motor centres.

If the influence of the vaso-constrictor centres of the oblongata is excluded by section of the cord in the cervical region, changes are produced by injection similar to those obtained in the intact animal.

When blood containing pituitary extract is artificially circulated through the vessels of a limb isolated from the rest of the circulatory system, vaso-constriction is obtained, succeeded by dilatation when blood free from extract is passed through the vessels.

The active substance exerts a stimulating influence upon the centres of the vagus, producing slowing of the pulse, but as this is also observed when the vagi have been cut, it is probable that there is a direct action upon the ganglia and muscle fibres of the heart. The extract acts although the vagi are paralysed by atropine, and the effect is not abolished by section of the depressors.

The response of the vagi and depressors to electrical stimulation is not abolished by the action of the extract.

When the depressors are excited during the period of action of pituitary extract a conflict arises between vaso-dilatation and vaso-constriction. The vessel musculature remains, although the extract is acting, under the influence of the vaso-motor nerves and the action of the latter may modify or abolish the effect of the extract.

W. A. JOLLY.

PATHOLOGY.

THE SEQUEL TO ASEPTIC LESIONS OF THE BRAIN. (Sur (565) les faits qui se développent à la suite des blessures aseptiques du cerveau.) SALA, *Arch. ital. de Biol.*, Tome 69, 1908, p. 1.

INCISIONS in the cerebral substance were carried out aseptically in a series of young dogs, cats, and rabbits, and the cicatricial tissue, stained by Cajal's method, was examined histologically forty-six hours, five days, and fourteen days after operation. The author regards his results as closely resembling the appearances presented

during regeneration of a divided nerve, but reserves his decision as to whether we have yet sufficient proof of a true regeneration.

There are present forty-six hours after operation numerous nerve rings, fine nerve fibrils terminating in closed rings, round, oval, or button-shaped.

After five days there are observed a certain number of rounded or spindle-shaped cells with granular protoplasm and large nuclei. There are also many nerve fibrils running alone or in small bundles, with a straight or tortuous course. On the borders of the incision lie rings and knob-like formations similar to those appearing after peripheral lesions. From the knob or bud an extremely fine fibre may occasionally be seen to pass out.

After fourteen days the cicatrix is found to be invaded by bundles of fine nerve fibrils arranged in a plexiform manner, with divisions and intercrossings. These are more numerous and closer around the vessels. Some of the fibrils present terminal enlargements. At points along the margins of the wound lie groups of rounded, oval, or pear-shaped bodies of homogeneous or reticulated structure. These are always in relation to fairly coarse nerve fibres, some lying at the extremity of a fibre, some on its course; others give origin to fine fibrils.

The pyramidal cells in the neighbourhood of the incision were examined forty-six hours after incision. The first part of the axis cylinder exhibits a club-shaped swelling, staining deeply, and of finely fibrillated structure. The part below this enlargement, lying adjacent to the wound, is ribbon-shaped, homogeneous, and stains light yellow. The appearance resembles that observed in the central stump of a divided nerve.

W. A. JOLLY.

**POSTERIOR COLUMN DEGENERATIONS FOLLOWING INJURY
(566) TO THE POSTERIOR ROOTS OF THE 7th CERVICAL
NERVES.** H. W. MITCHELL and A. M. BARRETT, *Journ. of
Nerv. and Ment. Dis.*, Oct. 1908, p. 545.

THE case is one in which the arches of the 5th cervical vertebra were fractured as the result of a fall. Death occurred a fortnight later. The 7th cervical segment was severely injured. The posterior columns, however, appear to have largely escaped, but nearly all the posterior fibres of the entering posterior roots at this level were blackened by the Marchi method, and were traceable up to the top of the cord as a compact bundle in the posterior column on either side. Descending degeneration in the comma tract was traceable down to the 6th dorsal segment.

EDWIN BRAMWELL.

CLINICAL NEUROLOGY.

INVESTIGATIONS ON THE CONVERGENCE REACTION WITH
(567) **REFLEX IMMOBILITY OF THE PUPIL.** (Untersuchungen
über die Convergenzreaktion bei reflektorischer Pupillenstarre.)
LACHMUND (Münster), *Berl. klin. Woch.*, July 1908.

DR LACHMUND cites the case of a female servant, aged 28, who had been an inmate of the asylum at Münster for seven months on account of hallucinations, etc.

The condition of the eyes was as follows:—The right pupil fairly wide, larger than the left, irregular, not reacting to light either consensually or directly. In a dim light the pupils were of equal size. The left pupil reacted promptly to light. Both pupils reacted promptly to convergence, but the right remained always a little larger than the left, and after contraction was not quite round. Accommodation good in both eyes. Vision good, fields good, muscular movements unimpaired, fundi normal, colour vision good. Examination of other cranial nerves and the whole nervous system gave negative findings. There was no evidence of syphilitic infection, and the patient had never suffered from any eye affection.

Dr Lachmund raises the question as to whether the right eye afforded a true instance of one-sided reflex immobility of the pupil, or whether the convergence reaction was also partly affected, as the right pupil on convergence remained a little larger than the left. Seeking further light on this question Dr Lachmund selected twenty-seven cases of general paralysis from the material of the asylum. He examined them as to the following points:—Whether the convergence reaction was plainly present, whether it was prompt and large in amount, whether both pupils after contraction were equally large, and whether they were round or misshapen. Among the twenty-seven cases he found twenty cases in which both pupils failed to react to light. In two cases there was only a trace of reaction, in two a good reaction, and in four the right and left pupils reacted differently. In sixteen cases the convergence reaction was present, while the light reaction was lost. In all these cases the contraction was prompt, though sometimes owing to the mental condition it was difficult to get the patient to converge properly. In twelve of the cases the pupils when contracted during convergence were unequal. In some of the cases the difference was very great. Further, out of the thirty-two pupils, twenty-nine were not round when contracted—namely, in thirteen pairs of eyes and in three single eyes. Again, it was

noted that among the pairs of eyes which showed unequal pupils after contraction eleven showed a difference also before the convergence reaction. Only in one case in which the pupils were equal before contraction did a difference make its appearance afterwards. This persistent difference in the pupils even when contracted Dr Lachmund does not ascribe to any defect in the convergence reaction, but simply to the fact that the convergence reaction though actively present is not able to make equally small those pupils which were unequal before the contraction started. In the case cited Dr Lachmund thinks that the pupil anomaly was probably congenital.

J. V. PATERSON.

A COMPARISON BETWEEN THE CLINICAL APPEARANCES (568) IN NUCLEAR AND TRUNK LESIONS OF THE VAGUS RECURRENT AND THE OCULOMOTOR NERVES. (Ein Vergleich der klinischen Erscheinungen bei Kern und Stamm-lähmungen des Vagus-Recurrrens und des Oculomotorius.) O. KÖRNER, *Ztschr. f. Ohrenheilk.*, 1908, Bd. 56, S. 153.

KÖRNER showed in 1894 that in an otitic temporo-sphenoidal abscess sufficiently large to compress the oculomotor nerve, the levator palpebræ superioris, or the sphincter iridis, or both, were first paralysed, while the remainder of the muscles supplied by the nerve were affected later or not at all. Albert Knapp's observations in the case of tumours confirm this.

This compares exactly with lesions in the trunk of the vagus nerve; there the abductors are first affected.

In nuclear lesions of the oculomotor nerve there is no such selective paralysis observed, and this raises the question, Is Semon's law correct in the case of nuclear lesions of the vagus?

Kussmaul and Cahn have shown that this law cannot be extended so as to include such lesions, and Cahn has shown that the paralysis of the vagus in tabetics (which Semon looks on as supporting his theory) is due to a neuritis of the nerve trunk and is not due to a nuclear lesion.

W. G. PORTER.

CEREBRAL COMPLICATIONS OF NASAL ORIGIN. (Über die (569) rhinogenen Gehirnkomplicationen.) ÓNODI (Budapesth), *Wien. Med. Woch.*, No. 33, 1908.

THIS paper commences with an account of the relations of the frontal, ethmoidal, and sphenoidal sinuses to the frontal and temporal lobes of the brain; the sphenoidal sinus may also come into relationship with the pons. Inflammatory conditions of these

accessory sinuses may give rise to intracranial complications, especially if the organism causing the sinusitis be very virulent. There is a direct communication between the veins of the sinuses and the meningeal network of veins, and an indirect connection through the diploic veins with the venous system in the dura mater. Statistics as to the frequency of sinus suppuration at post-mortem examinations vary very greatly, *e.g.* from 2 per cent. (Lichtwitz) to 50 per cent. (Martin). The cerebral complications of nasal origin are extra- and intra-dural abscess, meningitis, septic sinus thrombosis, and abscess of the brain. Wertheim has recorded 127 cases of intracranial suppuration out of 10,394 post-mortems, but only 14 of these were of nasal origin. The statistics as to cure of cerebral abscess of nasal origin are not favourable (Dreyfus, 2 cures out of 30 cases; Onodi, 7 out of 73 cases). Attention is called to the fact that brain abscess in these regions may remain latent, and a list is given of the symptoms without revealing any new or interesting points. Onodi remarks that at operation the posterior or cerebral wall of the frontal sinus may be intact, and yet at post-mortem an extra-dural or frontal abscess may be found. If indications of intracranial complication exist, the cranial cavity should be explored, even in the absence of necrosis of the posterior wall of the frontal sinus. Exploratory puncture of the brain here, as elsewhere, gives unreliable results, but the author is in favour of this proceeding. If, on exposing the dura, it appears tense and non-pulsating, it should be opened; but if the dura is normal in appearance and pulsates, the question of further operation depends on the operator himself and on the symptoms, because cerebral abscess may be present although the dura shows pulsation. The rest of the paper, which is not very interesting, is occupied with statistics as to distance of the lateral ventricles from the frontal sinus and other regions.

J. S. FRASER.

A CASE OF SEVERE VERTIGO AND TINNITUS; DESTRUCTION OF THE LABYRINTH; CURE. MACLEOD YEARSLEY, *Lancet*, Sept. 19, 1908.

LAKE and Milligan were the first to treat "unbearable" vertigo and tinnitus by operation on the labyrinth: the present case brings the number of these operations up to nine. The patient, a man of 47, had had otorrhoea in childhood, and since then had been somewhat deaf; for twelve months before operation he had not been able to hear his own voice. His first attack of vertigo occurred twelve years ago, and for the last three years he had continuous roaring noises in the ears and frequent attacks of giddiness.

There was a history of vertigo and deafness in the family, and the patient himself was very constipated. Drug treatment had been tried and found to be useless. The noises in the ear were always worse before an attack of giddiness. Turning the head quickly to one side or looking up brought on an attack, during which external objects appeared to move to the right, but the patient himself felt that he would fall to the left. The tympanic membranes were indrawn, and naso-pharyngeal catarrh was present. Tuning-forks were not heard by bone conduction, the left ear was quite deaf, and the right ear could only hear the Edelmänn-Galton whistle up to 4000 D.V.S. C_{512} , C_{1024} , C_{2048} were heard for a short time by air conduction. With feet together and eyes closed the patient swayed to the left; nystagmus was produced by rotation.

It is a pity that there is no note of the results obtained by syringing the ears with hot and cold lotion. As nystagmus was produced by rotation, the patient must have had at least one functioning vestibular apparatus.

Yearsley at first prescribed hydrobromic acid and quinine, and inserted a seton in the neck; in spite of change of air, the vertigo and tinnitus became "unbearable," and operation was agreed to. On completion of the radical mastoid operation, the left cochlea was opened and cleared out through the inner wall of the tympanic cavity. Above and behind the facial nerve the external and posterior semi-circular canals were opened and followed to the vestibule, which was scraped with a fine spoon. As a result of this operation there was a left-sided facial paralysis, but in a few months this almost entirely passed off. Immediately after the operation there was nystagmus, vomiting, and tinnitus for a few days, but these also improved, so that in ten days the patient was able to walk downstairs. Three months later the patient was quite free from giddiness and nystagmus, but had slight tinnitus after taking tea or coffee. He had become an adept at lip-reading, though he could hear the ordinary voice by means of an ear-trumpet applied to the right ear.

J. S. FRASER.

ACUTE GONORRHOEAL INFLAMMATION OF THE LABYRINTH.

(571) (*Labyrinthite blennorrhagique aiguë double.*) A. HÉBERT, *Bull. de la Soc. de méd. de Rouen*, 1907, p. 117.

A MAN, aged 20, who had suffered from aural trouble for several years, contracted gonorrhœa in November 1905. Examination of the ears at this time showed tubal obstruction and deafness, which under appropriate treatment considerably improved. On December 25 he developed epididymitis. On the same day he became completely deaf, and suffered from vertigo, tinnitus, and

staggering. Nystagmus was present, and was most marked on looking to the right. No tubal obstruction was found, and the diagnosis, subsequently confirmed by Lermoyez, of gonorrhœal inflammation of the labyrinth was made. Lumbar puncture, in which 18 c.c. of cerebro-spinal fluid were removed, was followed by disappearance of the vertigo and titubation and diminution of the tinnitus, but the deafness persisted unchanged in spite of pilocarpine injections, which were continued for over a month.

J. D. ROLLESTON.

A FURTHER CONTRIBUTION TO THE HERPETIC INFLAMMATIONS OF THE GENICULATE GANGLION, etc. J. RAMSAY HUNT, *Am. J. of Med. Sci.*, Aug. 1908, p. 226.

IN this paper the author elaborates in detail the syndrome previously described by him, of which the distinctive features are herpes zoster oticus, facialis or occipitocollaris, with facial palsy and auditory symptoms. There are various clinical types which may be fused into a single large group. The underlying pathology is a specific inflammation of ganglia of the spinal (unipolar) type, including, in addition to the geniculate, the ganglia of the acoustic nerve and possibly those of the glossopharyngeal and vagus. Emphasis is put on the fact that mild inflammatory reactions may be present in ganglia above or below the chief focus, producing pains but not eruptions in their zones. He regards the gasserian, geniculate, acoustic, glossopharyngeal, vagus, second, third, and fourth cervical ganglia, as embryologically, histologically, and clinically a series or chain (the acoustic only differing in having bipolar cells), and concerned in the production of the syndrome. A hæmorrhagic inflammation in any one will be followed by the usual manifestations of herpes zoster, the eruption being on its respective zone. The term *herpes oticus* should be confined to those cases where the eruption is confined to the cone-shaped zoster zone of the geniculate ganglion (the tympanum, auditory canal, concha, and an adjacent marginal area on the external surface of the auricle). The other features of the syndrome come from the proximity of the geniculate ganglion to the facial and terminal divisions of the auditory nerve, the inflammation extending to these by contiguity of structure, the effects being enhanced by the enclosure of these structures in a common sheath, situated in a narrow osseous canal. Hence the facial palsy and symptoms referable to the auditory nerve, which are of two types — hypo-acousis merely, or symptoms resembling Ménière's disease, tinnitus, deafness, vertigo, vomiting, nystagmus, and disturbances of equilibrium.

J. H. HARVEY PIRIE.

GNORRHOEAL NEURITIS OF THE AUDITORY NERVE. (*Neuritis* (573) *acústica gonocóccica*.) P. J. MARTINO, *Rev. Méd. del Uruguay*, March 1908, p. 66.

A MAN, aged 25, who had suffered two years previously from gonorrhœa, followed by orchitis, and had had a second attack four months ago, was suddenly affected by buzzing in the right ear a month after leaving off urethral injections. Shortly afterwards both ears became affected. Subsequently complete deafness in the left ear developed. Examination showed the existence of a bilateral otitis interna, the external and middle ear being quite normal. The only focus of infection was the persistent urethral discharge, in which gonococci were still present. Under appropriate treatment the urethritis was cured, and the patient concurrently noted a diminution of the noises in his right ear, though the deafness in the left persisted without modification. Small doses of potassium iodide and electrical treatment were adopted, and complete recovery took place within one and a half months. Martino regards the case as one of auditory neuritis, which started with a period of irritation and ended in paralysis of the nerve, and thinks that a large number of cases of obscure deafness may be due to gonorrhœal infection.

J. D. ROLLESTON.

SCARLATINAL RHEUMATISM. (*Zur Frage vom Scharlach-* (574) *rheumatismus*.) G. E. WLADIMIROFF, *Archiv f. Kinderheilk.*, Bd. 48, 1908, p. 214.

WLADIMIROFF thinks that two different affections are included under the denomination of scarlatinal rheumatism. Whereas in some cases the complication appears as a serous synovitis in which the morbid process is exclusively located in the joint, in others it develops as a scarlatinal neuritis. In such cases there is no swelling of the joint. Though the pain is situated chiefly in the joint, it is not confined thereto, but is felt throughout the limb, and varies in character and intensity, as is usually the case in neuritis. Wladimiroff examined the superficial peroneal nerves and one of the dorsal digital nerves of the foot in two children of seven and nine years, who died in the second and third week of scarlet fever, after suffering from rheumatoid pains in the legs, and found the characteristic lesions of all stages of neuritis present. Wladimiroff thinks that in further investigations attention should be paid to changes in the other nerves, especially the vagi and phrenics, and that if similar changes are found in these nerves in scarlet fever to those described by him in diphtheria, more light will be thrown

on those cases which die within the first few days of the disease in which macroscopical data are often insufficient to explain the cause of death.

J. D. ROLLESTON.

SEROUS SPINAL MENINGITIS. (*Zur Kenntniss der Meningitis (575) serosa spinalis.*) KURT MENDEL and S. ADLER, *Berl. klin. Woch.*, 1908, p. 1596.

MENDEL was consulted by a woman, aged 36, in whom spastic paraplegia with exaggerated reflexes, positive Babinski, Oppenheim and Mendel-Bechterew signs, sensory disturbances and sphincter troubles pointed to a spinal cord affection, while tenderness over the spines of the second, third and fourth thoracic vertebræ indicated the situation of the affection. The predominance of the pain in the right side of the back and chest and of the motor weakness in the right leg suggested that the right half of the cord was chiefly compressed. A history of lung trouble of ten years' duration pointed to tuberculous disease of the vertebræ as the cause of the compression. On the other hand, the good state of nutrition, the absence of deformity, pyrexia, cough or sputum, and a negative ophthalmo-reaction rendered this doubtful. Finally the diagnosis was made of an extra-medullary tumour of unknown nature, situated at the level of the second and third thoracic vertebræ. In favour of this view was the fact that at the first examination Brown-Séquard's syndrome was present, which was consequently replaced by the condition just described.

Laminectomy was performed by Adler. The spines and laminae of the second to fourth thoracic vertebræ were removed. The bones and periosteum showed no pathological change. No tumour was found. The normal pulsation of the cord was absent. The dura was opened, and some scanty fluid escaped under slight pressure. The arachnoid was then punctured, and about one and a half drachms of clear fluid spirted out. The cord, which now began to pulsate regularly, showed nothing abnormal beyond a slight depression and a milky opacity of the arachnoid. The operation was followed by slow but decided improvement. Five months later the patient was able to stand and walk a few steps without support. The sphincter troubles had entirely disappeared, and the sensory disturbances were much less. The reflexes were now abnormal only on the right side.

In the present case the cause of the meningitis was not ascertained. In most of the published cases disease of the vertebræ or of the cord was present. In his review of the literature Adler shows that circumscribed spinal meningitis is by no means rare. Its tendency to simulate a tumour of the spinal

cord is illustrated by the fact that out of twenty-two laminectomies performed by Krause for the removal of a supposed tumour, a localised serous meningitis was found to be the essential, if not the only, cause of the paralytic phenomena in six cases.

J. D. ROLLESTON.

SERODIAGNOSIS IN PSYCHIATRY AND NEUROLOGY. (*Die* (576) *Serodiagnostik in der Psychiatrie und Neurologie.*) STERTZ, *Allg. Ztschr. f. Psychiat. u. psychisch-gericht. Med.*, Bd. lxxv., 1908, p. 565.

EMPLOYING Wasserman's serodiagnostic method in 111 cases, Stertz obtained a positive reaction only in such conditions as were associated with syphilis. Forty-five cases of general paralysis were tested by this method, the cerebro-spinal fluid giving a positive reaction in 88·8 per cent., and the cerebro-spinal fluid or blood in 95·5 per cent. The cerebro-spinal fluid was examined in five cases of tabes: three gave a positive reaction. In eight cases of syphilis of the central nervous system, the cerebro-spinal fluid gave a uniformly negative reaction, but the blood serum, examined in five of those cases, gave a positive reaction in two. Seven cases of late latent or cured syphilis were tested. The cerebro-spinal fluid was negative in all; the blood serum was once positive and once negative. In the remaining forty-six cases (cerebral tumour, tuberculous meningitis, alcoholism, hydrocephalus, myelitis, multiple sclerosis, etc.) the reactions both of the cerebro-spinal fluid and of the blood serum were constantly negative.

W. T. RITCHIE.

BIOLOGICAL STUDY OF THE CEREBRO-SPINAL FLUID IN (577) **ANTERIOR POLIOMYELITIS.** MARTHA WOLLSTEIN, *Journ. of Exper. Med.*, July 8, 1908, p. 476.

THIS is the record of an attempt to find evidence of a micro-parasitic origin of the disease by the employment of the biological reaction of complement deviation, according to the methods of Bordet and of Wassermann and Bruck. The cerebro-spinal fluid was employed as a probable container of the hypothetical antigen. The fluid was obtained by lumbar puncture from twenty different cases of varying age and stage of the disease. The results obtained show, in the words of the author, that no two interacting substances, presumably antigen and antibody, capable of uniting and anchoring complement were demonstrable in the blood serum, cerebro-spinal

fluid, and organ-extracts studied. Therefore the diagnosis of poliomyelitis by means of a serum reaction is apparently not possible, and no light could be thrown on the etiology of the disease by this reaction.

J. H. HARVEY PIRIE.

LARYNGEAL CRISES AND PARESIS OF THE ABDUCTORS OF (578) THE VOCAL CORDS AS IMPORTANT EARLY SYMPTOMS OF TABES, WITH THE REPORT OF A CASE. OTTO T. FREER, *Journ. Amer. Med. Assoc.*, 1908, Vol. xli., p. 815.

THE patient, a man æt. 37, had been ill for six months. The illness began with attacks of vomiting. Three months ago he had a sudden catch in his breath; two days later he had an attack of spasmodic stoppage of breathing which began with a cough which was arrested by a closure of the throat so forcible that he could not breathe, and fell to the ground unconscious. He had had many similar attacks since then, but has not again become unconscious. On laryngeal examination, faulty abduction was observed of both cords. Neurological examination showed typical signs of tabes. There is sometimes a tickling sensation in the larynx before an attack, and during the seizure there is severe pain in the larynx. The number of attacks varies greatly; there have been as many as seven in one day, and sometimes several weeks pass without one occurring.

W. G. PORTER.

A CASE OF CERVICO-BULBAR SYRINGOMYELIA, COMMENC- (579) ING WITH HICCOUGH. (Un cas de syringomyélie cervico-bulbaire: début par un hoquet persistant.) SOLLIER and CHARTIER, *L'Encéphale*, Sept. 1908, p. 249.

SYMPTOMS of reflex irritation, such as nausea and vomiting, are rare in syringobulbia, though not unknown. Hiccough is even rarer. When it has been recorded it has appeared as a symptom late in the disease, and has been neither regular nor constant.

In the present case it was the first bulbar symptom, preceded solely by slight thermal hypæsthesia, of spinal origin; further, it persisted with great regularity, after a sudden onset. It is due in all probability to irritation of bulbar sensory nuclei.

S. A. K. WILSON.

SEGMENTAL HYPERTROPHY OF THE ARM IN SYRINGO-
(580) MYELIA. (*Hypertrophie segmentaire considérable du bras*
et de l'avant-bras avec dissociation syringomyélique des
sensibilités.) DESPLATS, *Nouv. Icon. de la Salpêtr.*, May-June
 1908, p. 200.

A CASE of syringomyelia, in which the right upper arm was eight centimetres more in circumference than the left, and the girth at the epicondyle six centimetres more, an increase which involved not merely the superficial structures but also the bone.

S. A. K. WILSON.

A CONTRIBUTION TO THE STUDY OF DISEASES OF THE
(581) CONUS MEDULLARIS. (*Beitrag zu den Erkrankungen des*
Conus Medullaris.) S. S. ROBINOWITSCH, *Berl. klin. Woch.*,
 Aug. 31, 1908.

THE author points out that disease of the conus medullaris (sacral segments 3, 4, 5), except of traumatic origin, is very rarely met with. As a rule the epiconus (4th lumbar to 2nd sacral segments) is involved in the cord lesion. He records a case which exhibited symptoms of an almost pure conus lesion. The patient—an engine-driver—after a night of exposure complained of paræsthesia in the perineal region and of slight pains at the bottom of his back. Next followed difficulty in passing his water and constipation. This shortly gave way to complete incontinence both of bladder and rectum, with loss of sexual power. There was no weakness in the legs at any time. When examined, besides the symptoms complained of, the author was able to demonstrate anæsthesia (complete) of urethral and rectal mucous membranes, and of the skin supplied by the 3rd, 4th, and 5th sacral segments. There was loss of the anal sphincter reflex. The right ankle jerk was lost, a fact which proved that the lesion was not strictly limited to the conus medullaris. The other reflexes were normal.

As syphilis could apparently be excluded with certainty, the author diagnosed a myelitis, due to cold, invading the conus medullaris and affecting the epiconus only to a very slight degree. He excludes a lesion of the cauda equina by the absence of pain at onset or through the course of the disease.

The patient was given a thorough course of iodide of potassium to no purpose, and after five months without any improvement taking place, a course of strychnine was adopted with local treatment by faradism to the rectal and urethral mucous membranes. Improvement began to occur almost at once and progressed till

the patient eventually made a good recovery. A stationary period, in this instance of five months, seems to be the rule in lesions of the conus. Eulenburg and Rosenthal have recorded cases where it persisted for two and four years respectively before improvement set in. The author lays considerable stress on the importance of local electrical treatment in such cases.

C. M. HINDS HOWELL.

ANOSMIA IN TEMPORO-SPHENOIDAL ABSCESS. (*Anosmie (582) bei Schlafenlappenabszess.*) BLOCH and HECHINGER, *Arch. f. Ohrenheilk.*, 1908, Bd. 76, S. 32.

ONLY two cases appear in the literature of anosmia in temporo-sphenoidal abscess, one reported by Stokes and the other by Habermann.

The authors have observed a third case (in 1902). A radical operation had been previously performed in 1901 on the right ear; in 1902 the same operation was performed on the left side. Five weeks later symptoms of cerebral abscess appeared. The temporo-sphenoidal lobe was explored and an abscess found. Three weeks later the sense of smell was tested and was found to be absent on the left side.

In July 1907 the patient could smell as well on the left side as on the right.

In Cases 1 and 3 the loss of smell was on the side of the lesion. In Case 2 it was on the opposite side. It would appear from these observations that the sense of smell should be tested in a case of suspected temporo-sphenoidal abscess.

W. G. PORTER.

APHASIA. REPORT OF TWO CASES. JOHN HAY, *Liverpool Med.-(583) Chir. Journ.*, Jan. 1908.

CASE I.—An example of the serious disturbance, including aphasia, which may occur as the result of a slight ocular defect, and in which relief is obtained when the error of refraction is corrected.

CASE II.—A man, aged 68, with arterio-sclerosis and high blood-pressure, was attacked by complete sensory and almost complete motor aphasia, unaccompanied by any paralysis or loss of consciousness. Two days after the onset of the aphasia some weakness was noted in the right hand. Three days later recovery was almost complete, and he was able to give an accurate and detailed account of the onset of the symptoms and of his condition during the continuance of the aphasia. The recovery was brief,

lasting about six days. Speech became again affected, and finally the aphasia, both sensory and motor, was complete: right hemiplegia developed, most marked in the arm and hand, which were flaccid and powerless; this was accompanied by right hemianæsthesia, especially noticeable in the hand and forearm.

The paralysis both of motion and sensation became worse, respiration became Cheyne-Stokes in character; he passed into a stuporose condition and died.

Cerebral thrombosis appears to be the likely explanation of the symptoms and course of the disease, but as no autopsy was permitted this supposition could not be verified.

The gradual onset, without any signs of cortical irritation or intra-cranial pressure, the temporary recovery, followed by the appearance and slow increase of the paralysis, and the peculiar and suggestive disposition and progression of the loss of power—for the lesion spread by stages corresponding to the areas supplied by the branches of the middle cerebral—all suggest thrombosis of these vessels.

The writer suggests that Broca's area had escaped, the motor and sensory aphasia being caused in the first instance by the involvement of the fourth division of the middle cerebral, by which means the integrity of the auditory and visual speech centres was destroyed.

The outcome of this was motor and sensory aphasia, uncomplicated by paralysis or loss of consciousness, but as the thrombosis spread down the vessel the motor cortex became involved.

AUTHOR'S ABSTRACT.

A CASE OF PURE APHEMIA (CORTICAL ANARTHRIA). LADAME (584) and VON MONAKOW, *L'Encéphale*, March 1908, p. 193.

THIS case formed the subject of a brief communication at the International Congress at Paris in 1900. It concerned a woman of fifty-five, who at the age of forty-five suffered a slight apoplectic attack, with transient right facio-brachial paresis, which was followed by absolute and complete mutism. The patient presented all the symptoms of so-called sub-cortical motor aphasia, without a trace of agraphia. Up to the time of her death, eleven years later, she was able to write spontaneously and correctly. In these eleven years she said "Yes" once, "No" once or twice; seven months after the attack she said "Good-bye, my little one"; two days later she said "Good-bye." With considerable effort she said "Thanks very much" once, but with these exceptions she remained dumb to the day of her death.

Her condition consisted of the following :—

- (a) Loss of spontaneous or voluntary speech.
- (b) Inability to repeat words.
- (c) Inability to read aloud.

On the positive side—

- (a) Complete integrity of “le langage intérieur.” No word-deafness or word-blindness.
- (b) Conservation of spontaneous writing.
- „ of copying.
- „ of writing to dictation.
- „ of the comprehension of words (spoken or read).
- „ of the power of reading to herself.

She was not completely aphonic ; she could emit a laryngeal sound which she was capable of modulating ; in chanting psalms in church she could make a rhythmical humming sound, her lips being closed. She died of diabetic coma in 1901.

At the autopsy an area of disease was found to occupy the foot of the third left frontal convolution and the lower part of the ascending frontal convolution, with the exception of a small section of the operculum covering the insula. The lesion was a hæmorrhagic cyst, with cicatricial thickening of the neuroglial wall. The left cortical centres for face, larynx, tongue, maxilla, hand and thumb were completely destroyed by the lesion. Yet clinically these structures were not paralysed.

A. Relation between the clinical symptoms and the anatomical lesions.—The area of disease involved association, commissural, and projection fibres of the white matter, as well as the cortex. But measurements show that more of the cortex was destroyed than of the white matter, so that the lesion may be fairly said to be cortical rather than sub-cortical, although, according to present views, the clinical picture was one of sub-cortical motor aphasia.

Examination revealed many cortical areas on the left side which were secondarily impaired as a result of degeneration attendant on the primary lesion. From this lesion of Broca and the operculum the *projection* fibres are completely lost as far as the bulbar nuclei. Although their number is relatively less than the loss of *association* fibres passing in all directions through the white matter, fibres both trans-cortical and sub-cortical, of varying lengths and of indeterminate origins, yet the sole clinical cerebral symptom was that of mutism.

There was unmistakable anatomical evidence of complete interruption of all fibres passing between Broca's area and the hypothetical centre for writing (the latter, indeed, was certainly involved in the lesion), as well as those to or from auditory and

visual centres. In spite of these lacunes, the patient was able to write perfectly well with the right hand, though a large portion of the projection and association fibres of the arm area in the ascending frontal had disappeared. These facts constitute the best proof that memory functions are not seriously impaired by purely local and unilateral lesions. There was not the slightest impairment of movement of the right hand, neither ataxia nor astereognosis.

The most remarkable feature in this connection, contrary to all current ideas on cortical localisation, is that the total destruction of the motor centres for tongue, larynx, jaw, palate, throat, mouth, and arm has produced neither paresis nor ataxia of these muscular distributions (*cf.* Munk's experiments on the extirpation of limited area of the cortex in monkeys).

The negative symptom of mutism which this patient showed is far from being the necessary consequence of the destructive lesion of Broca and the operculum. Von Monakow's explanation is that it is the result of a prolonged cortico-bulbar diaschisis (inability to overcome the difficulty of innervating the medullary centres). Yet in some cases of Broca's aphasia certain words return. Why there should have been none in this case is difficult of explanation, except perhaps as above. Ladame dissociates himself somewhat from von Monakow over this point.

B. Anatomical conclusions.—Thalamo-cortical fibres forming tracts in the corona radiata passing from the thalamus to the cortex (Broca and the operculum) have their origin in the lateral part of the median nucleus and in the ventral nuclei, but not in the median portion of the median nucleus nor in the lateral ventral nucleus. These fibres going to the cortex from the thalamus form the centripetal path for phonation.

The corticofugal path is in the present instance easily followed, a centrifugal path for phonation which in the internal capsule is mixed with fibres of the pyramidal tract, and passes down in the pons dorso-lateral to the latter. The course of the tract for phonation can be followed clearly, as there is no pyramidal degeneration.

This abstract can convey little idea of the great value of this unique case, in which various points are recorded for the first time in medical literature.

S. A. K. WILSON.

BILATERAL MOTOR APRAXIA, WITH RIGHT HEMIPARESIS (585) AND APRAXIA OF THE EYE MUSCLES. DENY and MAILLARD, *Société de psychiatrie*, Paris, July 16, 1908.

A CASE of arterio-sclerosis in a man of 55, with right hemiparesis but no aphasia or dysarthria, who understands perfectly

what is said to him and answers correctly. Apart from various apraxic defects in the performance of movements of expression, etc., the chief point of interest in the case is the condition of the eye muscles. During the examination the patient frequently made the remark, "I can't do what you want, because I can't see." Yet his visual acuity is normal and there is no hemianopia. In order to see, his eyes must turn, so to speak, automatically to the object presented to him. Otherwise he may pick up the wrong object several times in succession. If a newspaper or a book be given him he can read only those lines on which his eyes fall involuntarily, but he is voluntarily incapable of following a line, and therefore of reading a sentence. The defect is therefore not one of vision but of voluntary gaze. His eyes remain fixed when he is told to follow an object as it is moved in front of him, but there is no ocular paralysis, for if he fix his eyes his head can be moved about passively and the eyes move in the opposite direction to the head. Apraxia of the ocular musculature is rare, but has been previously described.

S. A. K. WILSON.

A CASE OF APRAXIA, WITH AUTOPSY. JOHN H. W. RHEIN, (586) *Journ. of Nerv. and Ment. Dis.*, Oct. 1908, p. 619.

THIS is an elaborate account of a case of apraxia. The literature is widely referred to in the discussion of the problems which the case presents. The author summarises the case as follows:—A man of 55; at the time of admission to the home he was blind; was totally unable to designate the position of his limbs; could not locate touch anywhere; could not recognise objects by the sense of touch; and his touch and temperature senses were imperfect in the left hand. The left hand, although capable of some reflex acts, could not be moved voluntarily. The right hand was apraxic, and apraxic phenomena were present in chewing and walking. The autopsy revealed the presence of degeneration of the white matter of the right occipital and parietal regions on the convexity and the posterior portion of the temporal lobe, the calcarine region remaining intact. The inferior longitudinal fasciculus and the optic radiations were degenerated on the right, and probably, though less markedly, on the left. On the left side there was degeneration in the occipital and temporal regions to a much less degree, leaving the median surface intact. The corpus callosum in its posterior portion was degenerated. Elsewhere the brain was apparently normal.

EDWIN BRAMWELL.

PRECOCIOUS HEMIPLEGIA IN SECONDARY SYPHILIS. (Con-
(587) tribution à l'étude de l'hémiplégie précoce à la période
secondaire de la syphilis.) G. DUTHEIL, *Thèses de Paris*,
1907-08, No. 336.

HEMIPLEGIA, which is essentially a phenomenon of secondary syphilis, often occurs eight, ten or fifteen months after the chancre, but may occur much earlier—*e.g.* two months (Mauriac), or fifty-six days (Laseigne). Dutheil records the case of a man, aged 43 years, who was admitted to hospital on December 2, 1907, with a generalised syphilitic eruption of a fortnight's duration. The chancre had been noted five weeks previously. A papulo-tubercular eruption testified to the precocious malignancy of his syphilis. Energetic treatment was therefore adopted, but after thirteen injections pronounced mercurial stomatitis developed, and treatment was suspended from December 15th to the 25th. Twenty-six days after admission and seventy days after the chancre drowsiness and hebetude developed. Lumbar puncture showed an excess of mononuclears. Eight days later right hemiplegia with Babinski's sign and motor aphasia occurred. Death took place on January 12, 1908. At the autopsy no lesion could be found to explain the cause of the hemiplegia.

J. D. ROLLESTON.

A CONTRIBUTION TO THE DISCUSSION ON EXOPHTHALMIC
(588) **GOITRE, WITH SPECIAL REFERENCE TO THE ANTI-**
THYROID TREATMENT. A. GORDON GULLAN, *Liv. Med.*
Chir. Journ., July 1908, p. 325.

THIS paper gives brief summaries of the author's experience with rodagen and thyroidectin in the treatment of exophthalmic goitre. Nine cases were treated by the former, of which one was cured, seven greatly improved, and one remained *in statu quo*. Five cases were treated with thyroidectin; all improved greatly (one after non-improvement by rodagen). The largest dose of rodagen employed was 3i four times a day, but the author thinks larger doses, given with caution, might have even a better effect.

J. H. HARVEY PIRIE.

EXAMINATION OF THE BLOOD IN EXOPHTHALMIC GOITRE

(589) (*Blut untersuchungen bei Morbus Basedowii mit Beiträgen zur Frühdiagnose und Theorie der Krankheit.*) THEODOR KOCHER, *Arch. f. klin. Chir.*, Bd. 87, H. 1, p. 131.

THIS paper consists of a record of blood examinations, in a large number of cases, of exophthalmic goitre and a general review of the latest views on the etiology of the disease.

Kocher had full blood examinations made in 106 cases. The red corpuscles, especially in young females, were frequently above five millions. The leucocytes were almost invariably reduced, in very many cases to about 5000, the lowest count being 3500. The reduction was almost entirely in the polymorphs, which in one case formed 35 per cent. instead 75 per cent. of the total number of leucocytes. The lymphocytes were proportionately and sometimes actually increased, in some cases forming 57 per cent. of the total. The eosinophils were frequently increased, sometimes to 15 per cent. of the total, but this was not a constant finding.

A marked lymphocytosis is a bad sign. Lymphocytosis without a general leucopænia is prognostically not such a bad sign as when it occurs along with leucopænia.

In early cases a lymphocytosis is present, but is not well marked.

Operation on the gland had a striking immediate effect on the blood counts. In one case, on the day following operation, the leucocytes rose from below normal to 12,900; the neutrophils rose from 42 per cent. to 89·2 per cent., while the lymphocytes fell from 48·0 per cent. to 2·7 per cent.

Some months after successful operation blood examination showed, as compared with the counts before operation, an increase in the total number of leucocytes, and a return to nearer the normal proportion of neutrophils to lymphocytes. The coagulation time of the blood is lengthened, and the viscosity of the blood is increased in this disease.

The lymphocytosis points to a chronic infection or toxæmia, and many of the symptoms of this disease resemble closely those seen in cases of lymphatic leucocythæmia.

Kocher decides that the disease is no etiological entity, but that it is of toxic nature, the toxin acting either directly on the gland or on the nervous system.

D. P. D. WILKIE.

**OSSEOUS PLAQUES OF THE PIA-ARACHNOID AND THEIR
(590) RELATION TO PAIN IN ACROMEGALY.** S. LEOPOLD,
Journ. of Nerv. and Ment. Dis., Sept. 1908, p. 552.

A CASE of acromegaly and one of arterio-capillary fibrosis are described in which these osseous plates were well marked. Sauton and State advanced the view in 1900 that these plates account for the pains in different parts of the body which occur as a common symptom in acromegaly. The author combats this view. The following are the conclusions arrived at:—

1. Osseous plates are frequently present in the pia-arachnoid.
2. They are found in many diseases, such as uræmia, tuberculosis, retrogressive conditions, etc.
3. Arterio-sclerosis seems to be the underlying factor in their causation.
4. The presence of these plaques upon the spinal pia in acromegaly does not explain the production of pain in that disease.
5. There is no definite pathology of the spinal cord in acromegaly.

EDWIN BRAMWELL.

PSEUDO-APPENDICITIS HYSTERICA. (Über Pseudo-Appendicitis
(591) *Hysterica*.) KARL URBAN, *Wien. med. Wchnsch.*, No. 35, 1908,
p. 1918.

By this term is meant a combination of the well-known signs of hysteria and the signs of appendicitis, with localised or diffuse peritonitis and high fever. The writer found records of twenty cases in the literature, and records one case of his own. This was a boy, 18 years of age, who was suddenly seized with acute pain in the right side of the abdomen and vomiting, which continued for twenty-four hours before his admission to hospital. On admission his tongue was dry, respiration shallow, abdomen slightly distended and rigid, especially the right rectus muscle, temperature 99·2, pulse 72. He complained of "unspeakable" pain, and his whole behaviour was somewhat theatrical.

He was treated with hot fomentations, and the pain subsided, though the tenderness, the exact site of which varied from day to day, remained. Three weeks later he had a rigor, the temperature rose to 104·4, pulse to 144, without there being much to be made out in the appendix region, and on the following day pulse and temperature were normal. Twelve days later another rigor with temperature of 104·2, pulse 84, the patient complaining of great abdominal pain in the appendix region, and entreating the surgeon

to operate. As on the following morning the temperature was still 104° F., laparotomy was performed. The appendix was found normal, but was removed. Six weeks after his discharge from hospital he returned complaining of pain over the left mastoid region and demanding operation, but nothing abnormal could be found. While in hospital he became maniacal, and had to be removed to an asylum.

The writer regards the rises of temperature as being of a purely hysterical nature, being, indeed, disturbances of the thermal centre comparable to those commonly seen in the motor and psychical centres in hysterical subjects.

D. P. D. WILKIE.

**A NEW SIGN FOR THE DETECTION OF MALINGERING AND
(592) FUNCTIONAL PARESIS OF THE LOWER EXTREMITIES.**

C. F. HOOVER, *J. Am. Med. Ass.*, Aug. 29, 1908, p. 746.

A NORMAL person, lying on a couch in the dorsal position, when asked to lift the right foot *up*, keeping the leg extended, presses the left heel *down* to get a point of opposition. This can be felt by the observer's hand placed under the tendo Achilles. A hemiplegic, if requested to lift the extended paretic leg, will be found to offer this opposition with the healthy limb, whether any voluntary muscular strength be exhibited or not on the affected side. In malingering or functional cases, however, when the apparently paretic limb is requested to be raised, there will be found to be no complementary opposition offered by the normal limb, provided the subject is unaware of the object of the examination. The author has observed and used this test on two malingerers and two hysterical cases and in a large number of hemiplegics.

J. H. HARVEY PIRIE.

**AN INTERESTING NERVOUS SYNDROME IN SECONDARY
(593) SYPHILIS. (Una interessante sindrome nervosa della sifilide
secondaria.)** G. BOSCHI, *Riforma medica*, 1908, p. 907.

BOSCHI records the case of a woman, aged 32, who for the past two years had been subject to insomnia, hysterical convulsive attacks, and psychical disturbances. Large doses of morphia and heroin only aggravated her symptoms. Other sedatives, and hypnotics such as veronal, and tonics proved of no avail. The simultaneous occurrence of osteocopic pains suggested specific

treatment. The hysterical symptoms soon disappeared, but the insomnia persisted in spite of thirty sublimate injections, and only yielded to salicylate of mercury.

J. D. ROLLESTON.

PERSISTENT HEREDITARY OEDEMA OF THE LOWER
(594) **EXTREMITIES.** (*Cedème persistant héréditaire des jambes, avec exacerbations aiguës.*) HOPE and FRENCH, *Nouv. Icon. de la Salpêtr.*, May-June 1908, p. 177.

A. W., aged 18, was only three months old when it was observed that her feet were swollen, without there being any apparent reason for this. This œdema never disappeared, but gradually crept up the legs, till at the age of eleven she was forced to wear bandages constantly to keep the œdema within bounds. In 1904, aged fourteen, she commenced to have a series of acute exacerbations of the condition, which usually lasted for some days. In these attacks there was a smart rise of temperature with shivering; the legs, specially the right, became intensely swollen and red, and exquisitely tender.

On coming under observation in 1906 the patient presented a condition of marked swelling of the legs, from Poupart's ligament to the toes, not involving the genitalia or spreading on to the abdomen. The swelling was not unlike elephantiasis. All the bony landmarks in the legs were completely obliterated; the skin was not discoloured; the œdema was more or less uniform, and prolonged pressure of the finger was requisite to produce a dimple. Evidently the subcutaneous connective tissue was greatly increased, although some infiltration too was present. Sensibility was unimpaired. The muscular weakness of the legs was probably mechanical in origin. The sphincters were normal, and the urine contained no abnormal constituent. Examination of the blood revealed no obvious deviation from the normal.

The family history presented remarkable features. Thirteen cases of the same condition were traced to have occurred in forty-two individuals of five generations. Some of these gave a history of acute exacerbations, identical with those of the patient. A consideration of these facts makes the diagnosis of chronic hereditary trophœdema or persistent hereditary œdema clear.

A lucid discussion of the various hypotheses capable of explaining the condition is closed by the authors declaring in favour of a vasomotor neurosis.

S. A. K. WILSON.

**HIGH INCIDENCE OF NERVOUS COMPLICATIONS IN A HOUSE
(595) EPIDEMIC OF DIPHTHERIA.** (Gehäufte Erkrankungen des Nervensystems bei einem Hausepidemie von Diphtherie.) W. FEILCHENFELD, *Deut. med. Woch.*, 1908, p. 1632.

EIGHT children whose ages ranged from three to thirteen years were attacked with diphtheria. One died on the sixth day of disease, and of the remainder, five had nervous complications. Two presented palatal and ocular palsies, three disturbance of cardiac innervation manifested by syncopal attacks, and in one paresis of the lower extremities occurred.

J. D. ROLLESTON.

PSYCHIATRY.

THE THYROID GLAND IN INSANITY. (La glande thyroïde chez (596) les aliénés.) RAMADIER and MARCHAND, *L'Encéphale*, Aug. 1908, p. 121.

No fewer than 278 thyroid glands were examined in patients dying in different French asylums (Villejuif, Rodez, Rennes, Blois).

(1) The weight of the thyroid gland presents no constant peculiarity in various mental diseases, apart from cretinism.

(2) Macroscopic lesions of the gland vary very much in the different asylums, but, again, present no constant peculiarity in relation to mental disease. The commonest change is the existence of colloid cysts.

(3) Microscopic lesions are, of course, very common. In old people they are practically constant, and this makes caution desirable in any statement as to their frequency among the insane part of the population.

In forty-eight cases only eight presented no abnormality. The lesions found were commonly diffuse sclerosis with atrophy of many of the vesicles; less commonly, parenchymatous or interstitial thyroiditis, with desquamation of the epithelial lining of the vesicles. The conclusion is that it is impossible to establish any constant relation between the thyroid change and the form of mental disease from which the patients suffered.

S. A. K. WILSON.

THE BULBO-CAVERNOSUS REFLEX IN DEMENTIA PRÆCOX.

(597) (*Il segno di Onanoff nei dementi precoci.*) F. VINCENZO,
Riforma medica, 1908, p. 876.

VINCENZO examined the bulbo-cavernosus reflex in thirty-three cases of dementia præcox whose ages ranged from nineteen to fifty-three years. In only one was the reflex normal, in sixteen it was very feeble, and in sixteen it was completely absent. The histories of these cases show that the sexual life of precocious demented is abnormal. All were celibates but three, and these were childless. The great majority were confirmed masturbators or perverts, while the rest were characterised by frigidity, torpor and mysticism. Corresponding to the functional disturbance Vincenzo found various anomalies of the sexual apparatus, such as deficient development or malformation of the penis, scarcity of pubic hair, hypo- or hyperæsthesia of the mucosa of the glans and loss of pulsation in the dorsal artery of the penis.

The writer concludes that Onanoff's reflex is of importance in the diagnosis and prognosis of the various forms of sexual neurasthenia, and that it is a valuable symptom in the study of dementia.

J. D. ROLLESTON.

A CASE OF DEMENTIA PRÆCOX (PARANOID FORM), WITH

(598) **AUTOPSY.** (*Un cas de démence précoce à forme paranoïde, avec autopsie et examen histologique.*) ANGLADE and JACQUIN,
L'Encéphale, June 1908, p. 453.

INFORMATION on the pathological anatomy of dementia præcox is so scanty and uncertain that the findings in this case are of interest.

Apart from certain chromatolytic and pigmentary changes in the cells of the cortex, changes the interpretation of which is always difficult, the authors found definite areas of subcortical sclerosis throughout the brain, which were easily identifiable both by touch and to the naked eye, and microscopical examination only served to render them more evident. They predominated in the ascending frontal, the frontal lobes, and the occipital lobes. They consisted of areas of intense gliosis in which were recognisable gliomatous elements, with a dense reticulum formed of large neuroglial fibres. They bear a certain resemblance to what is known as *l'état vermoulu* of senile brains. Whether the lesions described are to be considered the anatomical substratum of dementia præcox it would be premature to decide. The authors

suggest that there exists a diffuse sclerous sub-cortical encephalitis during the course of the disease which may become concentrated at certain points and lead to certain more or less focal symptoms, as existed in the case here reported.

S. A. K. WILSON.

APRAXIA AND DEMENTIA PRÆCOX. FROMARD, *L'Encéphale*, (599) Aug. 1908, p. 162.

Two cases of dementia præcox (it is, however, permissible to question the diagnosis on the facts supplied) presented more or less typical ideational apraxia. The patients, in the performance of somewhat complicated acts, "went off the rails" in various ways, either by short-circuiting (apraxia by anticipation), or by inattention (apraxia by suspension), or by mistakes of a "perseveration" type (apraxia by substitution), or by division of attention (apraxia by "intersion"). As an instance of the first of these, the patient was asked to pour water out of a carafe into a tumbler and drink it, but he drank out of the carafe instead; of the second, the patient was asked to make the sign of the cross, when he brought his finger to his forehead and then stopped altogether; of the third, he was asked to "put his fingers to his nose," when he brought his hand up to his nose and then began to scratch an eczematous area on the skin; of the fourth, the patient had a tumbler in his hand, which he was asked to put down, and he was then directed to close an envelope in his other hand; he put the envelope down, and proceeded to lick the edge of the glass with his tongue. A division of this kind, into apraxia by anticipation, substitution, suspension, or "intersion," can be simply one of convenience; it is obvious that the limits of each cannot be drawn rigorously.

S. A. K. WILSON.

JUVENILE GENERAL PARALYSIS, WITH APRAXIC SYMPTOMS. (600) CLAUDE and LEVI-VALEUSI, *Société de psychiatrie*, Paris, July 16, 1908.

THE patient is a girl of 22, with a definite syphilitic heredity and clear indications of the accuracy of the diagnosis. She presents apraxic phenomena in the shape of perseveration, motor apraxia, and ideational apraxia. Agnosia, however, cannot be excluded, which renders the clinical picture less instructive.

S. A. K. WILSON.

TREATMENT.

THE TREATMENT OF SPINA BIFIDA BY DRAINAGE OF THE
(601) **CEREBRAL SUBDURAL SPACE.** PETER PATERSON, *Lancet*,
Aug. 15, 1908, p. 456.

THE author's case was that of a dorsi-lumbar meningo-myelocoele in an infant two months old, associated with double congenital talipes equino varus, paresis of the lower limbs, and a mild degree of hydrocephalus. As the sac of the spina bifida was on the point of rupture it was excised, the spread-out cord and nerves separated with difficulty and returned inside the spinal groove, while the wound was firmly closed by suturing the edges of healthy skin round the base of the tumour. Shortly after this operation the fluid re-accumulated, the skin stretched, and cerebro-spinal fluid began to escape through the stitch-holes. In order to relieve the tension, the cerebral subdural space was drained into the tissues of the scalp by trephining over the right parietal bone, opening the dura mater with a crucial incision, and stitching the turned-back edges of the flap to the pericranium, the wound in the scalp being sutured and covered with collodion. The effect of this was to produce a very marked oedema of the scalp for some days, but also a great diminution in the tension of the swelling. Two months after this operation the hydrocephalus began to increase, in consequence of which drainage of the ventricles was performed. The author employed several silk threads, the inner ends of which were tied together and inserted into the ventricles, while the outer ends were spread out like a wick into the superficial tissues of the scalp. This was followed by oedema of the scalp, which lasted for ten days, and a gradual diminution in the size of the head. The fontanelles became lax and the parietal bones overlapped. Death, which took place three weeks after, was attributed to persistent vomiting, from which almost from birth the child had suffered. At the autopsy the opening made for draining the spina bifida was found to be closed, but the threads in the ventricles were draining satisfactorily. In summing up, the author states that in future he would utilise the threads to drain both the subdural space and the ventricles, as it would appear from this case that they were acting as a drain for the spina bifida by way of the subdural space, since

- the opening made for the former was found to be occluded.

C. B. PAUL.

CLINICAL STUDIES OF THE SURGERY OF OTOGENIC MEN-
(602) **INGITIS.** (*Klinische Studien zur Chirurgie der Otogenen Meningitis.*) G. ALEXANDER, *Arch. f. Ohrenheilk.*, 1908, Bd. 75, S. 222, and Bd. 76, S. 1.

THE author classifies cases of otitic meningitis as follows:—1. Meningitis in otherwise uncomplicated cases of acute middle ear suppuration; 2. meningitis in otherwise uncomplicated cases of chronic middle ear suppuration; 3. meningitis in cases of otitic brain abscess; 4. meningitis in cases of otitic thrombo-phlebitis and extradural abscess; 5. meningitis in cases of suppuration in the labyrinth.

Each group can be further divided into:—1. Those cases where there is a visible anatomical connection with the ear condition; 2. those cases where there is no such visible connection; 3. tuberculous meningitis. Thirteen illustrative cases are fully reported and discussed.

The value of lumbar puncture is discussed. Increased pressure of the cerebro-spinal fluid is certain evidence of meningitis; the colour of the fluid is also important. Of great diagnostic value is the appearance of coagulation in fluid which has stood for from three to twenty-four hours. Lumbar puncture gives exact evidence as to the state of the meninges, but it can never in itself form a contraindication to operation. The opinion that no operation should be performed where the cerebro-spinal fluid is turbid is now happily abandoned; that does not mean that every case of purulent meningitis is to be operated on, however far advanced, but that the question of operation must be determined from the clinical symptoms, deep coma, paralysis, and so on. It must always be borne in mind that a purulent fluid may be obtained in cases of brain abscess, suppuration in the labyrinth, or in sinus thrombosis.

As regards the operation, the first essential is the thorough clearing out of the disease in the ear, and in suppuration of the labyrinth wide opening of the vestibule and cochlea is indicated. The sinus and the dura of the middle and posterior cerebral fossæ must be widely exposed, several incisions may be made in the dura. After operation frequent changes must be made in the dressings, and repeated lumbar puncture is of value.

W. G. PORTER.

THE SURGICAL TREATMENT OF EXOPHTHALMIC GOITRE.
(603) (*Die chirurgische Behandlung des Basedow'schen Krankheit.*)
AUGUST HILDEBRANDT (Berlin), *Berl. klin. Wchnschr.*, July 20,
1908, S. 1362.

THE writer is strongly in favour of early operation in this disease, and brings forward evidence in support of this view. Nothing has yet been discovered which will influence the thyroid secretion, and although anti-thyroidin does good in some cases, it very frequently fails. The bad results with surgical treatment have all been in severe cases which have resisted prolonged medical treatment, and the mortality after operation is not greater than that from the disease itself. Out of 177 cases of resection of the thyroid gland for exophthalmic goitre, there were 57·6 per cent. cured, 26·5 per cent. improved, 2·2 per cent. unimproved, and 13·5 per cent. died. He considers a patient cured if he can resume his daily work and if the tachycardia disappears. In hospitals where early operation is the rule the death-rate is very low. Klemm in 32 cases had no deaths. Kocher in his last 52 cases had only one death. Although operation in early cases is associated with very little risk, the writer does not recommend it unless the case has resisted medical treatment for one month. The operation of choice is to remove one-half of the gland, and should this not cure the symptoms the arteries supplying the other half should be ligatured. In advanced and severe cases he strongly approves of Kocher's method of ligaturing both superior thyroid arteries as a preliminary operation to removal of one-half of the gland.

The operation should always be conducted under local anæsthesia, a 1 per cent. solution of cocaine being used. Convalescence is usually very slow, months, and sometimes even years, elapsing before the body is completely freed of the poison.

Complete rest for several months after operation is an essential in the after-treatment of all cases.

D. P. D. WILKIE.

POLYGLANDULAR SYNDROMES AND OPOTHERAPY. (Les
(604) *syndromes polyglandulaires et l'opothérapie associée.*) L.
RÉNON, *Jour. des Pract.*, July 25, 1908, p. 465.

EXPERIMENTAL work has shown the correlation of the different glands which possess an internal secretion. Thus Rénon and Delille found that repeated injections of pituitary extract into rabbits caused over-activity and hypertrophy of the suprarenals, while after injection of suprarenal extract the pituitary body appeared normal or showed over-activity without hypertrophy.

Ovarian extract caused considerable pituitary congestion, as a rule accompanied by glandular over-activity. In man, as in animals, infections and intoxications produce more or less marked alterations in the thyroid, suprarenals, and hypophysis. Rénon examined these organs in typhoid fever, pneumonia, acute and chronic tuberculosis and uræmia, and found very marked though variable changes. Thus a woman with tuberculosis of the right thumb and hallux showed a total caseous degeneration of the suprarenals, hypertrophy and sclerosis of the thyroid, hypertrophy and over-activity of the hypophysis, and bilateral ovarian sclerosis. In a case of pulmonary tuberculosis, which was much improved by suprarenal medication, there was very marked sclerosis of the suprarenals and thyroid and over-activity and hypertrophy of the hypophysis. Such findings show that polyglandular lesions are the rule and uniglandular lesions the exception. In clinical medicine, though the syndrome is polyglandular, the disturbance of function may be more marked, and appear earlier in one gland than in the rest. In a typical uniglandular syndrome, although the changes in one gland predominate, the changes in the other glands play a more or less prominent part. Thus myxœdema and exophthalmic goitre are both constantly attended with genital troubles, *e.g.* amenorrhœa or testicular atrophy. Genital disturbance is also constant in acromegaly and gigantism, and the association of acromegaly with myxœdema or with Graves' disease is relatively frequent. In many women ovarian insufficiency is associated with signs of hyper- or hypothyroidism, or both. A polyglandular syndrome is well illustrated in the different periods of a woman's life. Thus hypertrophy of the thyroid is normal during puberty, and is associated with ovarian ataxia. Acromegaly may also commence at this time. Affection of the suprarenals, thyroid, and hypophysis during pregnancy explains the tachycardia, sensations of heat, perspiration, and rise of blood pressure observed at this time. At the menopause ovarian insufficiency is often associated with other glandular symptoms. Thus most cases of obesity at the time are due to an association of thyroid and ovarian insufficiency, and involvement of the suprarenals and hypophysis is shown by hypertension and symptoms of acromegaly. Acute and chronic infections and intoxications affect the genital apparatus, especially of women, producing dysmenorrhœa, irregularity of the menses, and amenorrhœa. The activity of the thyroid may become excessive, as in tuberculosis and acute rheumatism. Insufficiency of the suprarenals and pituitary body (*v. Rev. of Neurol.*, 1907, pp. 324 and 719), either separately or in combination, may produce tachycardia, asthenia, insomnia, and psychical troubles. The pathogeny of certain dystrophic disorders becomes clearer when a systematic examination of the blood vascular

glands is made. Thus in a case of sclerodermia in a young woman who showed marked troubles of menstruation, with migraine, headache, and anorexia, the symptoms were due to a combined insufficiency of the thyroid and ovaries. In adiposis dolorosa, obesity, and some cases of acromegaly, thyro-ovarian insufficiency is also present, and the corresponding opotherapy is indicated. In a case of myasthenia gravis, in which the onset of the affection was definitely associated with menstrual disturbance, ovarian insufficiency was followed by signs of pituitary incompetence—viz., tachycardia, hypotension, and oliguria. The combined administration of the ovary and hypophysis caused these symptoms to disappear rapidly. A polyglandular syndrome also enters into the pathogeny of some cases of myopathy, neuro-fibromatosis, and paralysis agitans. In instituting treatment one should always start with uniglandular medication. In a polyglandular syndrome, in which the disturbance of a single gland predominates, corresponding uniglandular treatment will often suffice, since the exhibition of the extract of one gland often causes over-activity of the rest. When uniglandular treatment is insufficient, or produces no effect whatever, polyglandular treatment must be adopted, and, unless signs of intolerance develop, be continued for three weeks or a month, at the end of which time uniglandular opotherapy should be resumed. Glandular extracts must be employed with care, for their activity is considerable.

J. D. ROLLESTON.

Reviews

THE SIMULATION OF INSANITY. (*La simulation de la folie.*)

Professor A. MAIRET (Montpelier). Coulet et Fils, Montpelier, 1908.

IN this book, which is one of the first works wholly devoted to such a study, the subject of Simulated Insanity is treated of under four headings, viz., 1. Its Historical Aspect; 2. Its Clinical Aspect; 3. The Contrast between Simulated and Actual Insanity; 4. The Relation of Simulation to Responsibility.

Insanity has been feigned in all ages, and where history is available, as in the Bible and in the works of ancient Greek and Roman authors, instances are given to substantiate this assertion. David feigned insanity to gain the protection of Achish, King of Gath. Ulysses, Solon and Brutus also feigned insanity. To a study of the character of Hamlet, from the point of view of

simulated insanity, the author devotes twenty-four pages. He concludes that Hamlet is both a melancholic and a simulator, and that the portrayal of such states of mind is the result of an intimate knowledge by Shakespeare of the clinical aspects of insanity. To prove this, an interesting comparison is made between the first and second MSS. of *Hamlet*; or, as the author puts it, "The Shakespeare of about twenty-one years of age is compared with the Shakespeare of about thirty-six years of age." The younger Shakespeare knows practically nothing of insanity, the older Shakespeare has acquired a very intimate knowledge of it.

Hamlet, from a technical point of view, is considered a very bad simulator; his simulation is intermittent, and he puts too much spirit and depth into it.

Having considered feigned insanity in relation to general history, the author devotes a chapter to its history in medicine. The most important contribution to this subject is that of Zachias, who in 1688 wrote a chapter about it in his "Book of Medico-Legal Questions." Since the days of Zachias, the forms of insanity simulated have increased in number, as have the facilities of diagnosis, owing to a wider knowledge of the subject. The sources of inquiry indicated by Zachias are still the best modern elements for diagnosis.

The bulk of Prof. Mairer's book naturally deals with the clinical features and diagnosis of simulation. As a general rule, insanity is feigned for two main reasons: (1) To obtain an immediate personal satisfaction; and, in the majority of cases, (2) to escape punishment. Eighty-four per cent. of the cases exemplified had committed criminal acts. In this connection it is important to notice that it is not the gravity of the crime that induces simulation. Feigned insanity is rare, and the reason for this is the general repulsion that insanity inspires. The majority of prisoners prefer to pass as criminal rather than lunatic. The idea of simulating insanity does not usually originate in the mind of the simulator. It is generally suggested by a relative, a warder, or a fellow-prisoner. To simulate is very difficult, and all criminals are not capable of it. The simulator is usually a degenerate, in whom predisposition to insanity shows itself by moral perversion, and sometimes added to this, epilepsy or hysteria; or an arrest of development, more or less marked, is noticed, proceeding in some cases to imbecility. The simulator may, however, be very intelligent.

The forms of insanity simulated are dealt with in detail. They embrace mania, melancholia, stupor, delusions of persecution and grandeur, dementia, deaf-mutism and atypical forms; and in simulating these forms, not only their symptoms but also their evolution can be imitated.

The elements that raise doubts in the mind of the physician as to the genuineness of a case of insanity are the shades or faults in the picture when it is compared with that of actual insanity. The perfect simulator is rare. Where he exists, insanity has been copied as the result of association with, and the study of the insane.

In making a diagnosis, the author suggests an indirect and a direct examination. The indirect examination embraces an inquiry into the antecedents of the patient, the motives of any crime committed, the relation of the first appearance of signs of insanity to the crime, and the manner in which the mental troubles arose. In making an indirect examination, it is necessary to remember that parents, friends and neighbours may, by affection, interest, or pity exaggerate the mental state of the patient, or even falsely affirm the existence of a pathological state. Direct examination is to a great extent concerned in the inaccuracies of the clinical picture presented, by comparison with that of actual insanity. A precise knowledge of insanity and its several clinical forms is essential to the examiner. To obtain a correct diagnosis it is necessary not only to visit the patient frequently, but also to have the reports of persons accustomed to watch and record the course of insanity, and under whose care the examinee has been placed.

The elements which make for doubt as to the reality of the presence of insanity are brought prominently before the reader in concise paragraphs printed in italics, as follows:—

“When a prisoner, suspected of insanity, shows neither inheritance nor predisposition, doubt as to the existence of insanity in him should be entertained.”

“When a prisoner . . . has a history charged with crime, these doubts should again occur to the examiner.”

“The epoch of the appearance of mental troubles may have, in the discovery of feigned insanity, according to the case either no rôle, or a rôle more or less important—sometimes very important. The suddenness of the appearance of insanity ought always to raise doubts, more or less strong, according to the cases—weaker when the insanity takes the form of general delirium, considerable, and even of major importance, when there is the appearance of mental aberration with partial delirium.”

Complementary processes of diagnosis consist in attempting to elicit certain passions, which, if felt normally, would add to the suspicion that simulation is present.

In view of the proverb, “*In vino veritas*,” attempts have also been made to assist the diagnosis by putting the patient under the

influence of wine, opium or ether, and noting the mental results. It is important to have the avowal that the mental condition is simulated. This may be obtained by a ruse—by threats, pain, or punishment, such, for instance, as the use of the cephalic douche.

Feigned insanity is also found amongst the insane, but the author does not accept Greisinger's reason for it, viz., that it is done to satisfy some morbid pleasure. Prof. Mairet is of opinion that the insane simulator has a more practical end in view—like the criminal, *but not knowing of his insanity*, he wishes to escape punishment.

Insanity simulated by an insane person consists either of an exaggeration of his particular form of insanity, or of an entirely different form from that which already exists in him. The clinical phenomena in sane and insane simulators are alike.

The next part of Prof. Mairet's book is devoted to "alleged insanity," of which a prisoner, who alleges that he was in a state of dementia when he committed the criminal act, is invariably the subject. Two forms of "alleged insanity" are considered:—(1) The Amnesic Form, and (2) The Maniacal Form, resulting from alcohol, morphia, fear and impulse. Examples of "alleged insanity"—which is rare—are given in detail.

Finally, simulation and responsibility are considered. The author discusses whether simulation, as a blameable act, should be looked on as an aggravation of the crime, and the prisoner punished accordingly. He suggests that the mental condition of all simulators should be examined in order to find out whether they are not responsible.

This work, from beginning to end, sustains the interest of the reader. There are very few typographical errors, and much subject matter that is original. It will well repay study and perusal by all interested in mental problems.

HAMILTON C. MARR.

DISEASES OF THE SPINAL CORD. R. T. WILLIAMSON. Oxford Medical Publications. London: Henry Frowde and Hodder & Stoughton, 1908. 15s.

THIS work, as the author states in his preface, is based upon notes of lectures delivered at the Manchester Medical School during the last fifteen years. The notes have been largely added to, and the lecture-form has been altered. The aim of the work is to be a text-book, and an introduction to, rather than an exhaustive account of, the diseases of the spinal cord. The book, however, is no mere compilation. In every chapter there is abundant evidence of the author's independent judgment and original investigations. The

illustrations, of which there are 190, are with one exception made from the author's own drawings, photographs, and micro-photographs. The schematic drawings especially will prove of the utmost value to the student, as they tell their tale with singular clearness and are most suitably chosen to explain the text. The author's style is direct, clear, and attractive, and unessential details are not allowed to obscure the general perspective.

Students into whose hands the reviewer has placed the book are unanimous in their praise of the work in these respects, all agreeing also that the explanations and descriptions it contains are exactly what they need as an introduction to the subject.

With all this it should be further said that the author is fully abreast of all the modern work on the anatomy, pathology, and symptomatology of diseases of the spinal cord. There are six introductory chapters on the structure of the cord, general pathology, histology and functions of the cord, symptoms of spinal diseases, electrical and other modern methods of examination, and on the diagnosis and localisation of diseases of the cord. In his consideration of the individual diseases the writer has departed from the usual sequence, and has grouped them according to the predominance of some main symptom, such as symptoms of a transverse lesion of the cord, atrophic paralysis, spastic paralysis or paresis, ataxia, etc., and has given three additional chapters on spinal meningitis, spinal syphilis, and traumatic neuroses. This arrangement is obviously not a scientific one, and it may be doubted if its disadvantages do not outweigh its advantages, but, after all, it does not materially interfere with the description of the individual diseases. There is a final chapter on the histological methods which have been found most useful by the writer. Each chapter contains sufficient references to the bibliography.

The book has been admirably produced, the text and the illustrations being clearly printed. It would have been more satisfactory had the illustrations of transverse sections of the cord been printed on a uniform plan, with the anterior columns directed either upwards or downwards. Might we suggest that such a change might be made with advantage in the next edition, which, we feel confident, will be soon called for?

DISEASES OF THE NERVOUS SYSTEM. By H. CAMPBELL THOMSON. London, Paris, New York, Toronto, and Melbourne: Cassell & Co., Limited, 1908.

THIS work by Dr Campbell Thomson forms one of a series of medical publications by Cassell & Co. As the whole ground of neurology is covered in 467 octavo pages, the descriptions are

necessarily very concise. The author has, however, succeeded in producing a volume which, while free from all trace of redundancy, is yet full of facts, marshalled in such a manner as to be capable of ready absorption by the student of medicine, for whom it is primarily intended as an introductory text-book.

It contains 98 illustrations, largely borrowed from other works, but well chosen and well reproduced.

BOOKS AND PAMPHLETS RECEIVED.

Georg Merzbach. "Die krankhaften Erscheinungen des Geschlechtes." Alfred Hölder, Wien und Leipzig, 1909, M. 5.20.

Max Löwy. "Das Krankheitsbild der überwertigen Idee und die chronische Paranoia." *Verlag Lotos*, Prag, 1908.

Gilbert Ballet. "Neurasthenia." Translated by P. Campbell Smith. Henry Kimpton, London, and Alexander Stenhouse, Glasgow, 1908. 6s.

Charles S. Potts. "Nervous and Mental Diseases, for Students and Practitioners." 2nd Edition, revised and enlarged. Henry Kimpton, London, and Alexander Stenhouse, Glasgow, 1908. 12s. 6d.

François Moutier. "L'Aphasie." (*Gaz. des Hôp.*, sept. 1908.) Levé, Paris, 1908.

L. Dugas. "Une Théorie Nouvelle de l'Aphasie." (*Journ. de Psychol. norm. et pathol.*, No. 5, 1908.) Félix Alcan, Paris, 1908.

Max Löwy. "Die Aktionsgefühle : Ein Depersonalisationsfall als Beitrag zur Psychologie des Aktivitätsgefühles und des Persönlichkeitsbewusstseins." Bellmann, Prag, 1908.

Dercum. "The Supposed Evils of Expert Testimony." *N.Y. Med. Journ.*, July 25, 1908.

Näcke. "Über Familienmord durch Geistesranke." Marhold, Halle, 1908.

Wieg-Wickenthal. "Zur Klinik der Dementia praecox." Marhold, Halle, 1908. M. 3.

Dreyfus. "Über Nervöse Dyspepsie." Fischer, Jena, 1908. M. 2.50.

Sir James Sawyer. "Points of Practice in Maladies of the Heart." Cornish Brothers, Ltd., Birmingham, 1908.

Review of Neurology and Psychiatry

Original Articles

TUMOUR MALFORMATIONS OF THE CENTRAL NERVOUS SYSTEM.

By WILLIAM G. SPILLER, M.D.,
Professor of Neuropathology and Associate Professor of Neurology
in the University of Pennsylvania.

MALFORMATIONS of the central nervous system occurring as tumour-like structures are recorded in the literature, but the cases are not very numerous. Within the past few years two interesting examples of embryonic structural defect have come under my observation. In one of these a tumour was found in the cerebello-pontile angle; in the other, the tumour was at the lower end of the cord.

(a) *Malformation of the Cerebello-pontile Angle.*—In examining a brain in which a tumour having the appearance of an endothelioma, and growing from the region of the Gasserian ganglion, had been found, I observed a small flat growth about the size of a small bean, situated on the under surface of the right lateral lobe of the cerebellum, at the angle formed by the cerebellum, medulla oblongata, and pons. This tumour, when studied microscopically, gave the following findings:—

Though lying close upon the brain, it did not form an intimate part of its tissue. It was closely connected with the choroid plexus of the fourth ventricle, and in a few places was

not differentiated from the pia covering the cerebellum. The choroid plexus was almost everywhere distinct from the tumour, but in a few places it formed intimate union with it, and the tumour in small areas had a border of cells of the ependymal type. The groundwork of the tumour was a loose neuroglia, with irregular, short and rather massive bands of denser neuroglia of varying thickness running through it in all directions. In some areas the ground substance was denser than in others. The tumour contained numerous blood vessels. Scattered all through the tumour, without any definite arrangement, were nerve cells, round, elongated, or triangular, resembling in shape and size the cells of Betz in the paracentral lobule, or the cells of the spinal ganglia. Some of the cells appeared degenerated, had peripherally-placed nuclei and swollen cell-bodies, and contained few or no chromophilic elements. Others had chromophilic elements like those of the pyramidal cells of the motor cortex.

The tumour in places had numerous medullated nerve fibres, as shown by the Weigert hæmatoxylin stain. These were almost confined to the periphery, and in some portions were parallel with the border of the tumour, and in others radiated from the periphery a short distance toward the centre. Most of these fibres had a distinctly degenerated appearance. In some places they formed a meshwork.

The interpretation of this tumour was made easy by the excellent article by Kasimir v. Orzechowski.¹ This writer states that his finding seems to be the only one of malformation of the recessus lateralis reported in the literature. He believes that the so-called acusticus tumours, and other tumours of the cerebello-pontile angle, are probably remains of the wall of the lateral recess. The tumour that he describes was covered in places by an endothelial lining, and contained nerve cells and nerve fibres, and seems to have been similar to the malformation in my case.

The embryological malformations of this region, as v. Orzechowski suggests, are probably not so rare as appears at present, and when attention is directed to the subject, the reports of such conditions will doubtless become more numerous.

It is difficult to decide whether any relation existed in my case between the malformation of the cerebello-pontile angle and

¹ v. Orzechowski, *Obersteiner's Arbeiten*, vol. xiv., 1908.

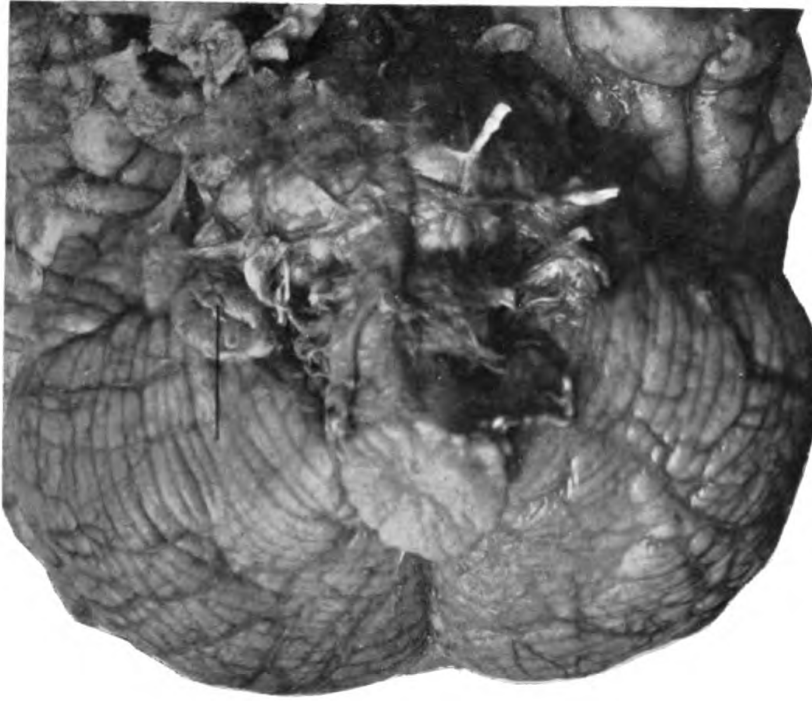


FIG. 1.



FIG. 2.

To illustrate Dr Spiller's Paper.

the tumour growing from the region of the Gasserian ganglion, but such connection is possible, inasmuch as Marchand holds that tumours of the Gasserian ganglion arise in the undifferentiated *Anlage* of the ganglion.

I have referred briefly to my findings in this case in a recent paper, but their importance seems to justify more consideration than was given to them there.¹

(b) *Malformation on the Sacral Region of the Cord.*—The case was one of extensive carcinoma, and has been reported without reference to the malformation of the spinal cord.²

The patient, a man, was admitted to the Philadelphia General Hospital, December 24, 1904. The face and upper limbs were not affected, but the lower limbs were almost completely paralysed, although some movement was possible at each hip and each knee, and the toes were moved slightly. The patellar reflex was exaggerated on each side, but ankle clonus was not obtainable. Babinski's reflex was distinctly present on each side. Sensations for touch and pain were preserved in the lower limbs, but tactile sensation was diminished on the soles of the feet. Retention of urine was present. There was only one record of pain, and that was made July 10, 1905.

He came again into my service in January 1906. At that time his condition was as follows:—He lay in bed with the thighs strongly flexed on the abdomen and the legs flexed on the thighs. He had slight voluntary power in flexion of each thigh, but it was very doubtful whether he had any voluntary movement of his toes. The slight upward movement of the toes which sometimes occurred was probably reflex. The lower limbs were much wasted. The patellar reflex and Achilles tendon reflex were absent on each side. The Babinski reflex was very typical on each side. Tactile and pain sensations were entirely lost in the lower limbs. He had no control of the urine or fæces, and the bowels did not move without enema. He moved the upper limbs freely, but the movements were weak. These limbs were also wasted. Biceps and triceps tendon reflexes were present on each side, and about normal,

¹ Marchand, "Festschrift für Rindfleisch," 1907, p. 265; Spiller, *American Journal of the Medical Sciences*, Nov. 1908.

² Spiller and Weisenburg, *Journal of Nervous and Mental Disease*, Aug. 1906; and *Weiner klinisch-therapeutische Wochenschrift*, Nos. 29, 30, and 31, 1906.

considering the general emaciation. The pupils were equal. The movements of the eyeballs probably were good, although it was impossible to get him to respond promptly. No impairment of cranial nerves was detected. The abdominal muscles were intensely rigid, and the abdomen was distended. A necropsy was obtained. Numerous carcinomata were found in various parts of the body.

A flat tumour was observed on the anterior part of the cord in the upper sacral region. It was about 2 cm. long \times 1.2 cm. broad, and was covered by the pia. Some of the nerve roots were implicated in the tumour. It was very friable.

The spinal ganglion of about the ninth thoracic root and this root also showed a little of the loose tissue seen in the tumour, but here, of course, it was outside the dura. This tissue contained osseous plates, here and there a few striated muscle fibres, fatty connective tissue, numerous vessels filled with red blood cells, and at one part a small mass of densely-packed round and elongated nuclei between which were connective tissue fibres. Between these various structures were loose bands of wavy connective tissue.

The tumour within the pia was of the structure described above. The bony plates stained purple, especially along the edges, with hæmalum, and contained cells separated from one another. Masses of cartilage-like tissue also were found. These stained very faintly, had a somewhat hyaloid appearance, and contained numerous cells with a large amount of protoplasm, much larger and very different from those within the bony plates. The muscle fibres were striated transversely and longitudinally, contained many elongated nuclei, and were like fully-developed muscle fibres. Here also were fat cells and some connective tissue fibres. Sections were taken from both the upper and lower ends of the tumour.

A spinal tumour containing striated muscle fibres resembling the findings in my case is described by J. Graham Forbes.¹ It was in the cervical region. The patient, a child aged five years and six months, had paralysis of both upper and lower limbs, supposed to be caused by cervical caries. He had always been "tottery" on his legs, had looseness of the bowels, and frequent and copious micturition. His head and shoulders drooped when

¹ Forbes, *St Bartholomew's Hospital Reports*, vol xli., 1905, p. 221.



FIG. 3.

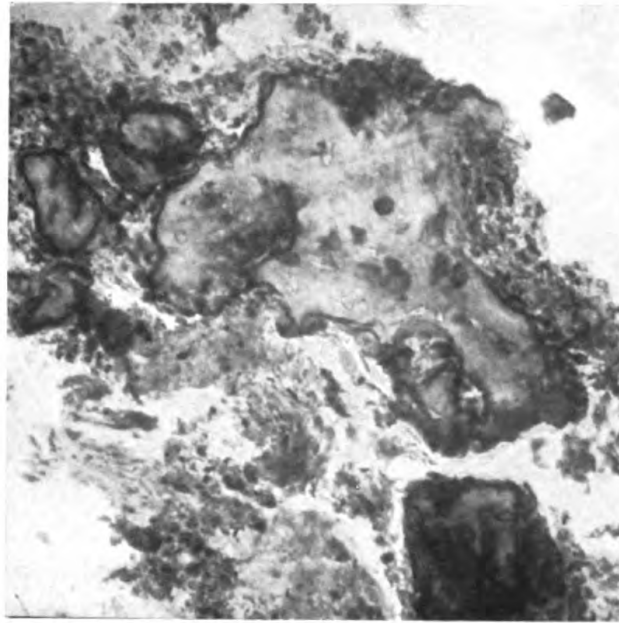


FIG. 4.



FIG. 5.

To illustrate Dr Spiller's Paper.

he was three years old. Later he had pain in the course of the posterior cervical nerves and rigidity of the neck. An operation was performed, an incision was made through the dura, and a growth about the size of a haricot bean protruded through the opening, and seemed to grow from the spinal cord. It was covered by the pia. The bulging portion of the tumour was removed.

The tumour on its posterior aspect was covered by a layer of dense fibrous tissue, probably thickened and adherent meninges. On the reverse side were many small strands of well-defined striated muscle fibres, portions of which were embryonic, and appeared as long fusiform cells with several nuclei arranged in columns toward the tapering end of the cell. (Such embryonic muscle cells were not found in my case.) These structures formed the most striking and characteristic feature of the growth. The strands of muscle fibres and cells were separated by broad bands of wavy fibrous tissue and small collections of fat cells. The centre of the tumour was occupied by poorly-staining connective tissue, interspersed with inflammatory cells and a large number of oval and round cells with fibrillary network, some of which possibly was neuroglia. In the anterior part of the growth was a cluster of large multinucleated giant cells resembling the myeloplaxes or osteoclasts of bone marrow, and apparently indicating the existence of young osseous tissue. The tumour was richly supplied with vessels. Many of the cells were free in the connective tissue; they stained poorly and showed an oval nucleus with a hyaline margin of protoplasm and shadowy ill-defined processes, and resembled degenerated nerve cells. The presence of fully-developed muscle fibre, with embryonic muscle cells and osteoclasts, showed that the growth was a teratoma, and on account of its vascularity it was regarded as a myo-angioma.

Forbes mentions a case of senile dementia described by Pick, in which bundles of smooth muscle fibres were found in the thickened membranes over the posterior surface of the cord. These muscle fibres were connected by strands with the hypertrophied muscle of the arterial walls.

Gowers'¹ case of a lipoma, with striated muscle fibres attached to the conus, is well known.

¹ Gowers, *Transactions of the Path. Soc. of London*, vol. xxvii., 1876.

682 TUMOUR MALFORMATIONS OF NERVOUS SYSTEM

I am indebted to Dr Alfred Reginald Allen for the photographs of the tumour on the conus medullaris.

LEGENDS.

- Fig. 1.—Photograph of the brain, showing the malformation of the cerebello-pontile angle, indicated by a line.
Fig. 2.—Photograph of the malformation on the sacral cord, indicated by a line.
Fig. 3.—Cartilage-like substance found within the tumour.
Fig. 4.—Bony plates within the tumour.
Fig. 5.—Striated muscle fibres within the tumour.

A RAPID METHOD FOR STAINING THE MYELINE IN NERVE FIBRES OF THE BRAIN AND SPINAL CORD (SIMPLE FORMOL OR FORMOL SULPHATE, FREEZING, ALUM-HÆMATEIN).

By Dr J. NAGEOTTE,
Physician to the Bicêtre Hospital, Paris.¹

THERE are numerous methods for the staining of healthy myeline. With the exception of Exner's method, which does not respond to all the requirements of pathological anatomy or give permanent preparations, these are all more or less derived from Weigert's method. The disadvantage which they all have in common is the necessity for a prolonged mordanting of the preparations. Weigert's rapid method does, it is true, give in a week excellent results as regards the cord, especially if the sections are treated with sulphate of copper, as I have indicated; but the sections are often friable, and the method does not apply to the brain.

The new method which I propose is based, like its predecessors, on the foundations established by Weigert, but in it the alum-lac of hæmatoxylin is used without previous mordant. By its means sections made by freezing pieces fixed in formol may be stained in a few seconds, and thus *we may have sections of nerves, of spinal cord, and of portions of the brain the day after autopsy, and large serial sections of the whole of the brain may be made, without embedding, eight to fifteen days later, if we*

¹ Communication made to the Société de Biologie, November 7, 1908.

have a suitable microtome. The necessity of complete fixation before freezing must not, however, be forgotten. As any method may be employed after formol, the technique of Nissl, Bielschowski, and others may be used for sections from the same block as those which are treated with the view of studying the myeline fibres.

I shall first describe the staining technique in itself, and then the modes of applying it to large sections of the brain.

Method.—For the nerves, spinal cord, and white matter of the brain, fixation may be carried out in a 10 per cent. solution of formol (containing 4 per cent. of formic aldehyde). But I have satisfied myself that the finest fibres of the cerebral cortex are affected by this solution, a fact which is of great importance in view of the almost general use of formol as a fixing agent. In order to stain the fibres of the cortex and to procure cortical plexus as rich as those given by Exner's method, we must add a corrective to the solution of formol. Starting from the idea that the density of the liquid might be a factor, I tried sulphate of soda and obtained excellent results by means of the following solution :—

Water	900
Formol	100
Sulphate of soda hydrated	10-70

This solution fixes the other elements as well as the simple solution of formol, and it does not prevent the employment of the methods of Nissl and Bielschowski. Strong solutions have the advantage of being slightly more dense than the brain, which at first floats and does not lose its shape, but they have the disadvantage of causing greater shrinkage and of fixing much more slowly than simple formol. I do not at present know exactly what proportion (perhaps variable) of sulphate of soda gives the best results, but I can affirm that the addition of this salt is useful. There is, therefore, every reason to use a sulphated solution instead of the ordinary solution for the formolisation of the cadaver, according to P. Marie's excellent method, and for the subsequent treatment of the pieces. Other salts should be tried and compared with sulphate of soda as modifications of formol solutions.

The sections, made by freezing, are placed in water. Before

staining, any fatty matter on the surface should be removed by alcohol. For this purpose it is sufficient to spread the sections out upon a plate and sprinkle them with alcohol. In this way their shape is not distorted. Prolonged immersion in alcohol does not affect the staining, but it has the disadvantage of contracting the sections, especially those of the cord. The finer the pieces are, the more easily is the fat removed from them.

Staining is carried out by means of the alum-lac of hæmatoxylin. The sections may be mordanted in alum and then treated by Weigert's hæmatoxylin solution, but by this method the fibres of the grey matter are badly stained. It is better to stain them directly in the hæmatein solution of P. Mayer. During the staining process the sections should be carefully spread out and should not cover each other. For this purpose spread the section out on a plate, drain it, and pour over it some drops of hæmatein. Then place the preparation in a warm moist chamber for half an hour. As regards sections of the cord, one can operate more rapidly by heating the preparation over a Bunsen burner till it begins to steam. The section will then be saturated in a minute. The hæmatein solution, which is poured off after staining, may be kept until it is exhausted.

After being washed in water the section is differentiated in a solution of Weigert's stain (ferricyanide, 2.5; borax, 2; water, 100), more or less diluted, then washed and mounted in the usual way. The sections must be well washed if the staining is not to become faint during mounting. It may be of advantage to add a trace of ammonia to the water in which they are washed. Pal's method does not give such good results.

Application to Large Sections of the Brain.—The brain, divided into segments one centimetre thick,¹ can be perfectly frozen and cut into sections. The most favourable temperature is between 1° and 2°. The sections are thoroughly flexible, and one may take them up with pincers without tearing them, and throw them into water. They may then be again caught with pincers or a needle, taken out of the water in a wrinkled condition, and the little bits of rag which they then look like are laid out on damp blotting paper and arranged in order.

¹ There are special microtomes for the purpose of making this division according to exactly parallel planes, so that the sections lost in this operation may be very few in number.

They will not be spoiled by being kept damp, and one can thus arrange them without any trouble in series, which may subsequently be stained. These manipulations are much more rapid than those necessitated by sections of pieces embedded in collodion and cut under the water.

To stain these sections, they are made to float in the water, which smoothes them out; they are then placed on a glass plate, the fatty matter on the surface thoroughly removed, and after having been sprinkled with absolute alcohol they are again made to float in the water. Then they are once more placed on a glass plate, drained, and after a thin ring of paraffin is traced round them by means of a hot iron, to keep it from running away, the staining fluid is poured over them. It should be noted that the sections become rigid and friable when they are fully stained, but as soon as they have been differentiated they regain their flexibility and may again be moved on the end of a needle from one receptacle into another.

ON THE ORIGIN OF THE FACIAL NERVE.

By ALEXANDER BRUCE, M.D., F.R.C.P.E., Physician to the Royal Infirmary, Edinburgh; and J. H. HARVEY PIRIE, M.D., B.Sc., Clinical Tutor in the Royal Infirmary, Edinburgh, Assistant to the Professor of Medical Jurisprudence, University of Edinburgh.

THE origin of the facial nerve has been the subject of much investigation and of many controversies. It is long since its alleged connection with the abducens nucleus has been disproved by Van Gudden and Gowers (1), and since Lockhart Clarke's (2) view as to the inferior facial nucleus has been set aside; but a number of other questions, which are of interest and importance from both the anatomical and the clinical standpoint, still remain to be settled. The object of the present paper is to endeavour to answer some of these, such as, for instance:—

- (1) Whether all its fibres arise from the well-known main nucleus in the pons?
- (2) Whether any of them have a crossed origin from the corresponding nucleus of the opposite side?
- (3) Whether there is a separate nucleus for the muscles of the upper face, viz., the orbicularis palpebrarum

frontalis, corrugator supercilii, and, if so, where is it situated?

- (4) Whether there is a separate nucleus for the muscles of the lips, and, if so, where is it situated?

The third and fourth questions have been raised by physicians in order to explain certain clinical phenomena which appear to be incompatible with the assumption that all the muscles of the face could be innervated from a common centre. The fact that in such diseases as bulbar paralysis and amyotrophic lateral sclerosis there is a comparative escape of the muscles of the upper face, while the muscles of the middle and of the lower face are profoundly involved, has suggested the possible existence of a nucleus for the nerves of supply of the upper face independent of the main nucleus of the facial nerve in the pons. On the other hand, the close association which exists between certain movements of the tongue and of the lips has led to the view that there must be a similarly close association of their nuclei of supply. Charcot (3) and Ross (4), following Lockhart Clarke (2), held that the muscles of the lips are innervated from an inferior facial nucleus, situated partly above the hypoglossal nucleus and partly also between it and the ependyma of the fourth ventricle; while Gowers (1) maintains that the centre for the lip muscles is in the upper extremity of the hypoglossal nucleus itself.

A. With regard to the nucleus for the upper facial muscles, Mendel (5), after section of the upper facial nerve in young rabbits and guinea-pigs, found atrophy of the cells in the lower part of the oculo-motor nucleus of the same side, and traced a connection to the facial nerve through the posterior longitudinal fasciculus. Mendel's belief that this group of cells constituted the nucleus for the upper facial nerve, apparently so completely solved the difficulty of the clinician that there has been a tendency to accept its existence as an independent nucleus without further sufficient proof. The remarkable immunity of the upper facial muscles in bulbar paralysis and in amyotrophic sclerosis, even where there is a high degree of paralysis of the lower face, has from the first been difficult to explain, and it has seemed essential to premise the existence of a nucleus altogether independent of the facial nucleus and escaping the degeneration

in which the latter is involved. Mendel's discovery seemed to supply the long-sought explanation. The clinician could now understand (or think he understood) why the nucleus of the facial nerve could suffer so severely and yet the middle and lower face alone be paralysed. The upper face escaped because its nucleus, far away from the main nucleus, was not involved in its degeneration.

There is reason to think, however, that the immunity of the upper facial muscles is not always so complete as from a mere examination of the position and movements of the eyelids it would appear to be. There is not infrequently a degree of paresis of the orbicularis with a greater or less dilatation of the palpebral aperture, and even when this dilatation is not apparent, there may be a considerable degree of paresis as evidenced by the reaction of degeneration in the muscles of the upper face. We have recently, in a case of amyotrophic lateral sclerosis, had occasion to demonstrate a reduction to a nearly equal degree of the electrical reactions in the upper and lower face muscles, in which there did not seem on inspection to be any paresis of the upper face; and we would draw attention in this connection to the condition of the eyelids and their electrical reaction noted in the remarkable case of diplegia facialis published in this review by Drs Fowler and Rainy (6), and which will be again referred to in another relation. They note that their patient, a child of ten months old, showed nothing particularly wrong with its eyelids before entering hospital; that during the few days of its stay in hospital before its death the lids could not be voluntarily shut very tightly, but that they covered the eyeballs fully during sleep. In spite of this the electrical responses were at first very feeble and subsequently absent altogether. The entire escape of the upper facial muscles is thus, in our opinion, more often apparent than real. Indeed, we venture to doubt if in any case in which there is advanced degeneration of the muscles of the lower face, the electrical examination of the muscles of the upper face will fail to show some degree of reaction of degeneration. It is also a remarkable fact that degenerations of the oculo-motor nuclei do not appear to be associated with paresis or paralysis of the upper face, as would almost certainly be frequently the case if the nucleus for the latter formed part of the oculo-motor nucleus, as stated by Mendel.

Wilbrand and Saenger (7), in their valuable work on the neurology of the eye, summarise the evidence in favour of Mendel's view. It is not very convincing.

(a) Obersteiner (8) found that after destruction of one oculo-motor root the posterior (or inferior) part of the corresponding oculo-motor nucleus was intact. It does not, however, necessarily follow that the part of the nucleus which has escaped is Mendel's centre for the upper face muscles. It is now a well-recognised fact that some of the fibres of the oculo-motor root have a crossed origin from the lower part of the nucleus of the other side. Therefore it is practically certain that division of one oculo-motor root would necessarily leave untouched those cells of the nucleus which are the centre for the fibres crossing from the undivided nerve of the other side. In support of this view we have the discovery by Siemerling and Boedeker (9) that a unilateral paralysis is followed by a degeneration in the anterior part of the nucleus of the same side, and by a bilateral paralysis in the distal—that is, the posterior or inferior part of the nucleus.

(b) The clinical evidence quoted by Wilbrand and Saenger (7) is as follows:—Hughlings Jackson described three cases of ophthalmoplegia externa associated with involvement of the orbicularis palpebrarum. Turner observed a case of nuclear ophthalmoplegia, with paresis of the upper facial muscles. Meyer has described an analogous case. Woods mentions a case of ophthalmoplegia externa with ptosis, in which there was a weakness of both orbiculares palpebrarum. Smith found in a case of total ophthalmoplegia a participation of the frontalis and orbicularis palpebrarum. Fuchs, in a case of bilateral ptosis, found also weakness of the frontalis muscles. Hanke noted in a case of bilateral ophthalmoplegia externa, which was either of congenital origin or dated from earliest infancy, that at a later period ptosis and paralysis of the frontalis appeared. Birdsall in a case of external ophthalmoplegia observed a diminution of the electrical excitability of the upper division of the facial nerve.

Such evidence, in the absence of control by post-mortem examination, must be regarded as merely suggestive. It is obvious that by the law of probability such associations of symptoms may occasionally arise without their being dependent on lesions of a single centre. There is nothing in the clinical

history of those cases to exclude multiple lesions either of similar or of different kinds; that is to say, there may have been more than one nuclear lesion or more than one peripheral lesion, or nuclear and peripheral nerve or muscle lesions may have been associated together.

On the other hand, Wilbrand and Saenger quote a case carefully examined by Cassirer and Schiff (10), in which, with *complete degeneration of the oculo-motor nucleus, even in its posterior segment, the function of the upper facial nerve was not in any way interfered with*, and they note, without, however, giving cases, that Bernhardt (11), Sauvigneau (12), and Siemerling (13), have stated categorically that they have found the upper facial nerve intact in nuclear ophthalmoplegia. Siemerling and Boedeker (9), after a series of exhaustive microscopical examinations of cases of progressive ophthalmoplegia, have arrived at the conclusion that there is *no evidence whatever that the oculo-motor nucleus has any connection with the muscles supplied by the upper facial*.

Wilbrand and Saenger, while admitting that the question of the upper facial nucleus is not yet settled, are inclined, on the strength of their own observations, to agree with Cassirer and Schiff, Siemerling and Boedeker, etc., in their opinion.

More recently the question has been attacked from another side—namely, from the study of the *reaction à distance* (or axonal reaction), which follows in the cells of origin of a nerve when its continuity is interrupted.¹

Marinesco (15) in 1898 divided the branch to the frontalis, orbicularis, and corrugator supercilii, and found a degeneration in the inferior part of the facial nucleus, and in the posterior part of its median nucleus. Section of the inferior facial produced degeneration in the external group of the facial nucleus. Complete division of the facial nerve caused axonal reaction in all the cells of the facial nucleus.²

¹ This method is calculated to give reliable data when the subsequent examination of the nerve centres is made at a sufficiently short interval after the initial section or destruction of the nerve. It is not of value, however, when an interval of many months, and still less of many years, has elapsed; therefore, such cases as Gianelli's (14), where the frontal branch of the facial nerve was divided fifty years before death, must be set aside.

² Marinesco has shown that the common facial nucleus varies in its outlines at different levels of its course, and slightly also according to the animal used. In the

Kotelewski (16), after experimental section of the upper branch of the facial, found degeneration in the dorso-lateral portion of the facial nucleus. This in all probability corresponds to the dorsal portion of the middle group of Marinesco, as the dorsal nucleus seems rather to lie behind the outer than the middle group (see Van Gehuchten's Fig. 487).

Parhon and Nadejdé (17) examined a case of cancer involving the superior facial, and found that what they term the first dorsal group¹ showed cells which were greatly atrophied. (The second and even the third dorsal group also showed less marked changes, which they regarded as due, in some measure at least, to lesions of nervous filaments which do not form part of the superior facial. They describe as belonging to the facial nucleus a group, also described by Wyrubow (18), situated between the common nucleus of this nerve and the nucleus of the sixth pair. They regard this group as the centre of the occipital muscle.)

Parhon and Minea (19) found that the nucleus of the upper facial nerve is represented by the first dorsal group of the nucleus of the seventh pair. They state categorically that the oculo-motor nuclei, including that portion which is situated in the depression of the posterior longitudinal fasciculus, do not send fibres into the trunk of the facial nerve.

All these observers, therefore, are unanimous that the origin of the upper facial is situated within the facial nucleus in the pons, and apparently in its dorsal part.

With regard to the origin of the nerve to the orbicularis oris, it is now universally admitted that the inferior facial nucleus of Lockhart Clarke (2) has no connection with the facial nerve; but the view of Gowers (1) as to the upper part of the hypoglossal nucleus constituting such a centre has met with wide acceptance. He has brought forward strong arguments in its support. He says: "The orbicularis oris and the transverse

middle of its length three cell groups—internal, middle, and external—can be distinguished. The middle group is again sub-divided into an anterior and posterior segment. At the upper and lower extremities of the nucleus the groups become reduced to one. (For illustration, see *Rev. Neurol.*, 1898, p. 30; also Van Gehuchten, *Anat. du Système Nerveux*, 4me ed., pp. 589 to 591; and Quain's "Anatomy," 11th ed., Vol. iii., Part i., p. 152.)

¹ The reference in the *Rev. Neurol.* does not give figures, so that it is not quite clear what the authors mean by the first, second, and third dorsal groups.

muscles of the front of the tongue have a functional relation closer, perhaps, than any other two muscles in the body equally distinct. Neither can be put in action without the other. The orbicularis is always involved in degenerative disease of the hypoglossal nucleus, and escapes in disease of the chief nucleus of the facial, as in a case of acute atrophic paralysis in which the face was involved." . . . "Considerable degeneration has been found in the fibres of the 'loop' of the facial nerve, even when the cells of the chief facial nucleus were but little affected—a mysterious fact. The posterior longitudinal fibres may be normal or partly degenerated (in proportion, it is said, to the affection of the hypoglossal nucleus); especially the longitudinal fibres suffer in the inner part of the reticular formation which are supposed to continue the anterior ground fibres of the cord. The degeneration is always bilateral."

Illustrations of the possible path from the hypothetical centre in the hypoglossal nucleus have been given by one of us (A. B.) (20), by Ferrier (21), and by Purves Stewart (22), who, in his valuable work on the "Diagnosis of Nervous Disease" states that the facial nerve arises from a nucleus in the lower part of the pons, but derives some of its fibres, namely, those for the orbicularis oculi, from the nucleus of the 3rd nerve, and others, namely, those for the orbicularis oris, from the hypoglossal nucleus.¹ On the other hand, however, Oppenheim (23), while figuring the connection from the 12th nucleus to the 8th nerve, regards it as being very improbable.

If it is the case that the nerve to the lower face arises from the hypoglossal nucleus, it must, as Gowers states, undergo degeneration in disease of the hypoglossal nucleus. A complete destruction of one hypoglossal nucleus should cause paralysis of the lips as well as the half of the tongue on the same side. The converse must also follow, that a section or destruction of the nerve of such a character as to produce *reaction à distance* in the main facial nucleus of the pons must also produce *reaction à distance* in that portion of the hypoglossal nucleus in which the lip muscle fibres take their origin. A case, there-

¹ Dr Purves Stewart, in the second edition of his book, which has just appeared, has modified this statement, saying that the cells for the orbicularis oculi extend as high as the nucleus of the third nerve, and those for the orbicularis oris as low as the hypoglossal nucleus.

fore, of complete facial paralysis in which there was total reaction of degeneration in all the muscles of the face would be, if examined sufficiently early, adapted to prove or disprove the alleged association. If it be found that the facial nucleus is alone involved, and the hypoglossal nucleus quite intact, that would be indisputable proof that the hypoglossal nucleus has no connection with the facial nerve. On the other hand, if the facial nucleus were degenerated, but a portion of the hypoglossal nucleus were also similarly affected (the hypoglossal nerve being, of course, in a normal condition), then the presumption would be very strong, or the proof even absolute, that the lip-facial muscles arose from the hypoglossal nucleus.

Similar reasoning would serve to prove or disprove the origin of the upper facial nerve from the oculo-motor nucleus.

The opportunities for clinical observation of such cases and their confirmation by autopsy are exceedingly rare, and therefore the question has remained for a long time in doubt; but we are in a position to describe a case which has been under the charge of one of us (A. B.) during life, and in which the medulla and pons have been examined microscopically by both of us after death, and which appears to us to justify the conclusion that the facial nerve has no origin in either the oculo-motor or the hypoglossal nucleus.

Clinical Case.—A. M., 65, harbour-master; recommended by Dr Dickson of South Queensferry; admitted to the Royal Infirmary, Edinburgh, in February 1908.

Abstract of Notes of History and Examination, as made by Drs Hugh More and W. Kelman Macdonald:—Sudden onset on 1st December 1907, with pains all over his body. Fourteen days later development of giddiness, which gradually increased, producing great uncertainty in walking, with fear of falling and tendency to sway towards the right side, without, however, any actual fall. Simultaneously there developed deafness, with buzzing noises in the left ear, the deafness in a short time becoming absolute. Also left facial paralysis. No headache. No other sensory or motor disturbances.

Previous history was satisfactory, except that his left eye was removed thirty years before for "some inflammatory trouble." Otherwise there was no personal or hereditary taint.

Examination of the face showed a complete left facial paralysis, the left forehead being smooth and immobile, the left

eyebrow lower than the right, and the aperture of the mouth having the characteristic obliquity. The mouth was somewhat drawn to the right side. The palpebral aperture, owing to the absence of the eye, was only slightly open. None of the muscles of the face were capable of any voluntary or reflex movement. There was no response to faradic stimulation. On galvanic stimulation of the muscles a slow and feeble contraction was obtained, but the cathodal closing contractions remained slightly stronger than the anodal closing contractions. The tongue was protruded without lateral deviation. The sense of taste was equally well conserved on both sides of the tongue. The examination of the ear by Dr Logan Turner showed no evidence of any middle-ear catarrh, past or present.

Closer examination of the nervous system showed nothing abnormal, with the exception of some defect of sight in the right eye, associated with an increased depth of the anterior chamber, tremulousness of the iris and hippus. There was no optic neuritis. The movements of the right eye were unimpaired. The stump of the excised eye-ball was capable of a little movement which was communicated to the eye-lids.

The superficial and deep reflexes were normal, the plantar reflex being of the flexor type on both sides.

Further examination revealed evidence of chronic tuberculosis in the upper part of both lungs, some dilatation of the heart, especially in its left ventricle, with a stenosis of the mitral orifice. The heart's action was somewhat irregular, the pulse tension low, the arteries tortuous and their walls thickened and beady. The urine showed a specific gravity of 1014, but it was otherwise normal. There was some evidence of rheumatoid arthritis in the hands, feet, and hip. There was also a reducible left femoral hernia and some tendency to varicose eczema in his lower limbs.

Diagnosis was made of neuritis of the facial and auditory nerve in the internal auditory meatus. The absence of evidence of middle-ear catarrh and the retention of the sense of taste and the association of deafness, giddiness, and facial paralysis indicated that the lesion was probably situated between the side of the pons and the bottom of the internal auditory meatus. The completeness of the facial paralysis was evidence of a total involvement of the facial nerve.

Subsequent Progress.—The patient, who was treated with rest and iodide of potassium, regained strength to a considerable extent, but did not recover from his giddiness, and died suddenly from heart failure on 2nd May 1908.

Autopsy.—The autopsy revealed general fibrous adhesions of pleura, chronic interstitial pneumonia in the upper halves of both lobes, the remainder of the lung showing chronic venous congestion with œdema. The heart was soft and flabby, with fatty infiltration, especially on its right side, dilatation of all its chambers without hypertrophy of the walls except to a slight degree in the left auricle. The mitral valve was dilated, with some thickening of its cusps. The tricuspid valve was also dilated. There was diffuse epicardial thickening, with localised “milk-spots” towards the apex of the left ventricle anteriorly and posteriorly over the lines of the vessels. There was extensive fatty infiltration of the wall of the greatly dilated right ventricle. Both coronary arteries were thickened and showed atheromatous patches. The aortic cusps were dilated, with thickening along their attachments and towards the corpora Arantii. The left ventricular wall was thin, friable, soft and fatty, the papillary muscles being fibrous towards their tips. The liver and kidneys showed a marked degree of chronic venous congestion, the kidneys also showing atrophy of the cortex, increase of pelvic fat, and some chronic interstitial nephritis.

The left optic nerve was atrophied, and the left 3rd, 4th, and 6th cranial nerves were somewhat thinned. There was some slight milky thickening of the arachnoid over the inferior surface of the cerebellum and between the lobes; also to a slight extent over the circle of Willis, and extending $1\frac{1}{4}$ inches in front of the optic chiasma to near the tips of both temporal lobes.

The brain was carefully hardened in formalin. The pons and medulla were embedded in paraffin, and serial sections were cut of the whole length of the facial, oculo-motor, and hypoglossal nuclei. These were stained throughout with Unna's polychrome blue and by Van Gieson's method.

Within the pons the facial nucleus of the same side was completely affected; not a single cell was found to be normal. The nucleus itself showed a considerable diminution in the number

of its cells. Those that remained had all reached the stage of atrophy, still showing a well-marked axonal reaction, with eccentric nuclei, chromatolysis, and more or less convex outline. Many of them also contained large masses of yellowish pigment granules.¹

On the other hand, there was no degeneration whatever in the facial nucleus of the opposite side. Its cells were without exception healthy. Many of them contained some yellow pigment, but apart from that, the Nissl's granules and the position and character of the nuclei were quite normal. It is hardly possible to conceive a more positive demonstration that none of the fibres of the facial nerve arise from the nucleus of the opposite side. Had it been otherwise we should have found normal cells in the degenerated nucleus, as well as degenerated cells in the opposite nucleus.

A similar examination of the hypoglossal nuclei demonstrated that their component cells were throughout quite intact. Here and there a cell showed some slight degree of pigmentation, but that was perfectly explainable by the age of the individual. Apart from that, the cells were well formed, their Nissl's granules everywhere presented their normal characters, and the nucleus occupied its normal position.

On examining the oculo-motor nuclei, no distinction could be made out between the two sides. Notwithstanding the removal of the eye-ball, and the resulting comparative inaction of the muscles, there was no indication of any change in the character of the cells on the affected side. Their nuclei were invariably central, their form normal, and their Nissl's granules stained with great distinctness. The nuclei on both sides were searched from end to end, and in no part was there any evidence of degeneration. The sections were submitted to several other workers in the Neurological Laboratory, and they likewise failed to find any difference between the two sides.

We might again refer to Drs Fowler and Rainy's (6) case of cerebral diplegia, in which they found, in what was practically a complete bilateral facial paralysis, that there

¹ Almost midway between the main nucleus and the abducens nucleus a small group of motor cells, described by Wyrubow (18), was found to be atrophied on the affected side and normal on the other. They thus evidently belong to the facial nerve.

was no abnormal change in either the oculo-motor or the hypoglossal nucleus, and no degeneration in the posterior longitudinal fasciculus.

Conclusions :—

1. That the upper facial nerve does not arise from the oculo-motor nucleus.
2. That the lip-facial fibres do not arise from the hypoglossal nucleus.
3. That there is no crossed origin of the facial nerve from the main nucleus.
4. That no crossed origin for any of the fibres has yet been discovered.
5. That all the fibres of the facial nerve arise from the groups of cells in the pons which lie behind the superior olive, and are known generally as its main nucleus—these groups being regarded as including the small accessory group situated a little behind it (Wyrubow), *i.e.* nearer to the abducens nucleus.
6. That the upper facial nerve probably arises from the dorsal part of the nucleus.
7. That further localisation of function of the nucleus has not yet been clearly established.

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- See also Alexander Bruce. "Contribution to the Question of the Origin of the Facial Nerve," *Scot. Med. and Surg. Journ.*, Nov. 1898. Tsuchida, "Über die Ursprungskerne der Augenbewegungsnerven, etc." (*Arb. a. d. hirnanatom. Institut. in Zürich*, H. 2, 1908).

Abstracts

ANATOMY

DESCRIPTIONS OF THREE CHINESE BRAINS. (Presented by (605) Dr F. W. Mott, F.R.S., to the Museum of the Royal College of Surgeons.) Part II. E. H. J. SCHUSTER, *Journ. Anat. and Physiol.*, Oct. 1908, p. 59.

THIS is a description of Brain No. II. The principal fissures are first described, and there follow (*a*) the principal sulci, and (*b*) the gyri and the remaining sulci in each of the lobes of both hemispheres. The position, length, and characters of the fissures; the position, shape, and dimensions of the gyri, and the peculiarities of both are given. Photographs of the various aspects of both hemispheres, with outline tracings of sulci and gyri, accompany the text.

E. B. JAMIESON.

BRAIN MATTER. (Über die Hirnmaterie.) M. REICHARDT, (606) *Monatsschr. f. Psychiatr. u. Neur.*, Oct. 1908, Bd. 24, S. 285.

AFTER deploring the disappointing results of brain histology in psychiatry, the author discusses at great length the possibility of discovering physical differences in the brain by non-microscopical methods, in particular by careful weighing. He summarises in this preliminary communication the various problems he has set himself to study by this means.

ERNEST JONES.

THE ANATOMY OF THE PROJECTION FIBRES OF THE OCCIPITAL LOBE. (607) **PITAL LOBE.** (Zur Anatomie der Projektions- und Balkenstrahlung des Hinterhauptlappens sowie des Cingulum.) VAN VALKENBURG, *Monatsschr. f. Psychiatr. u. Neur.*, Oct. 1908, Bd. 24, S. 320.

THIS is a careful description of a case of thrombotic softening, with a detailed consideration of the lessons taught by the succeeding degeneration. The author concludes that the geniculocortical radiation in its occipital portion is confined to the inferior longitudinal bundle. In contradistinction from Beevor, he maintains that the lateral portion of the cingulum is occupied by fibres having a fronto-caudal course.

ERNEST JONES.

ON THE FIRST PHASES OF THE DEVELOPMENT OF THE NERVE CENTRES IN THE VERTEBRATES. (608) **NERVE CENTRES IN THE VERTEBRATES.** (Sulle prime fasi dello sviluppo dei centri nervosi nei vertebrati.) V. BIANCHI, *Ann. di Neurol.*, Fasc. 1-2, 1907.

THE existence of spongioblasts, first shown by Golgi and His, has since been confirmed in the work of many writers. Opinions have differed as to the origin and mode of growth of what has been generally recognised as a supporting tissue.

The author deals with experiments performed by himself on the developing chick. He finds that the osmium bromide, the osmium bichromate, and osmic acid solutions, are greatly superior to all others. He obtained the best results with safranin, magenta red, and magdala red.

Two types of cells in the elements which form the medullary canal were distinguished in the earliest stages of development—the one consisting of elongated cells in contact with the internal and external surfaces of the tube, and the other of round cells with little protoplasm round the nucleus. The former he calls, with His, spongioblasts; the latter, neuroblasts.

As regards the position of the spongioblasts—many are situated under the meningeal surface, and send inward a long process that loses itself on the surface of the central canal, and a short prolongation to the external surface that frequently branches on reaching the meningeal surface.

According to the author the neuroblasts, besides becoming transformed into nerve cells, and contributing to the formation of the neuroglia, undergo frequently karyolysis. This phenomenon

may be witnessed on a large scale throughout the nervous system, and is to be regarded as a process of selection. It is possible that this material arising from nucleolysis may be utilised by other elements which go to constitute the nervous system.

F. GOLLA.

**THE CUTANEOUS BRANCHES OF THE POSTERIOR PRIMARY
(609) DIVISIONS OF THE SPINAL NERVES AND THEIR DIS-
TRIBUTION IN THE SKIN. HENRY M. JOHNSTON (Dublin),
Journ. Anat. and Physiol., Oct. 1908.**

THE writer of the paper has made a number of complete dissections of these nerves, and draws conclusions based upon his work regarding their origin, course, and distribution in the skin. He shows that contrary to what is generally stated a variable number of *external* branches from these nerves arising high up in the thoracic region succeed in reaching the skin. Though small in size, these filaments may travel a very long distance through the deep structures before becoming cutaneous, and supplying an area of skin which lies to the outer side of, or sometimes below, the area supplied by the internal branch from the same spinal nerve, yet is contiguous with it. The long course which the cutaneous nerves of the back take amid the muscles and fascia is clearly shown in the plates accompanying the paper, this course being in all cases more or less curved. The remarkable plexus on the back of the sacrum formed by the posterior primary divisions in this region is described.

It is interesting to compare the figures given to illustrate the areas of skin supplied by the individual branches of the posterior primary divisions of the spinal nerves as determined by dissection with those obtained clinically. A comparison with Head's figures shows that there is considerable agreement. Owing, however, to variations in origin in the lower cervical and upper thoracic regions, and to the free communication found between the nerves in their deep course, such pictures showing the areas of skin distribution obtained by clinical and dissecting-room methods must of necessity show some differences. The tracing of fine nerve fibrils into the skin presents obvious technical difficulties which one can hardly hope to entirely overcome. The paper is illustrated by drawings in the text and three plates in colour.

AUTHOR'S ABSTRACT.

**A METHOD FOR THE RAPID AND EASY DEMONSTRATION
(610) OF THE INTERNAL RETICULAR APPARATUS OF
NERVE CELLS.** (Une méthode pour la prompte et facile
démonstration de l'appareil réticulaire interne des cellules
nerveuses.) GOLGI, *Arch. ital. de Biol.*, T. 49, F. 2, Sept. 1908,
p. 269.

THE peculiarity of the structure in nerve cells, which the writer has described as "the internal reticular apparatus," has not received the attention it deserves, because, till now, there has not been a method to demonstrate it rapidly and correctly. Many writers still regard it as hypothetical. Others identify it with different structures, which have nothing in common with it. Holmgreen has identified it with structures which he describes under the name of "trophosphages." And still more remarkable, Cajal has identified it also with Holmgreen's trophosphages, referring to these two different organisations by the name of the "Reticular Apparatus of Golgi-Holmgreen." Others have followed in the same line.

Apart from the fundamental difference between the interpretation which Golgi has given to his reticular apparatus and that given by Holmgreen to his trophosphages, a glance at the figures of the two writers will exclude the possibility of identification.

With the method which Golgi now gives, this structure is revealed in almost all the ganglion cells of the nervous system (specially the intervertebral ganglia) hitherto studied.

The method is merely a modification of the photographic one used by Cajal and others. The most important point is the fixation of pieces—both as regards composition of the fixing fluid and duration of immersion in it.

1. Fixing mixture—Formaline (20 per cent. solution), 30 gr.
Saturated sol. of arsenic acid (very pure,
about 1 per cent.), 30 gr.
Alcohol of 96°, 30 gr.

Leave in this six to twenty-four hours.

Best results in pieces left in solution six to eight hours.

When fixing prolonged, one finds gradual modifications of the reaction, up to cessation of all elective reaction on reticular apparatus.

2. Pass into 1 per cent. solution of nitrate of silver.

Leave in this one to three hours. Longer immersion, even for several days, does not prevent good results.

3. Wash rapidly in distilled water.

Then develop in usual photographic solution (Golgi uses hydroquinone, 20 gr.; sulphate of soda, 5; formalin, 50; water, 1000).

In a few minutes superficial parts show reaction; in some hours development will have taken place through all the portions of tissue.

4. Wash pieces again with distilled water; harden in alcohol; this may be rapidly done.

5. Plunge into solution prepared at the moment, by mixing the two following:—*Sol. A.*—Hyposulphite of soda, 30 gr.; sulphocyanide of ammonium, 30 gr.; water, 1000 gr. *Sol. B.*—Chloride of gold, 1 gr.; water, 100 gr.

Sections, whether embedded in paraffin or celloidin, may be left exposed to action of this bath (in a watch glass) for a number of minutes, which we cannot pre-determine, for there are marked differences in different cases; the process should be watched, and stopped when sections have a marked grey colour.

6. Following operations are important, because they give more relief to various parts of tissue and demonstrate them distinctly:—

(a) Wash repeatedly in distilled water.

(b) Pass rapidly into following solution—

Permanganate of potassium, 0.50 gr.

Sulphuric acid, 1 gr.

Distilled water, 1000 gr.

(This operation should be watched, so that decolorisation is not excessive.)

(c) Wash rapidly, first in 1 per cent. solution of oxalic acid, then in distilled water.

(d) Stain with alum-carmin, and then wash.

(e) Pass through alcohol and mount in balsam.

ALEXANDER BRUCE.

CONTRIBUTION TO THE STUDY OF THE CELLS OF THE LOCUS

(611) OCERULUS AND SUBSTANTIA NIGRA. (Beitrag zum Studium der Zellen des Locus coeruleus und der Substantia Nigra.) GIUSEPPE CALLIGARIS, *Monatsschr. f. Psychiatr. u. Neur.*, Oct. 1908, Bd. 24, S. 339.

AFTER a brief description of the common yellow pseudo-pigment, the author describes the rarer, black, true pigment. His contrast between the two deserves quotation:—

Yellow Pigment.

1. Begins usually, at the sixth year, in the spinal ganglia. The time of appearance is inconstant.
2. Found in all nerve cells (rarest in Purkinje's). Shows great individual variation.

Black Pigment.

- | |
|---|
| <ol style="list-style-type: none"> Appears constantly in the first year, first in the locus coeruleus. Found exclusively in certain areas (locus coeruleus, substantia nigra, vagus nucleus, spinal ganglia). Similar in all individuals. |
|---|

Yellow Pigment.

3. Increases with age, and is related to various pathological processes (poliomyelitis, paralysis, etc.).
4. At times gives fat reaction.
5. Manifold staining reactions.
6. Also found in animals.
7. Single foci are small, spherical, and yellowish.

Black Pigment.

- Does not increase with age, and is not related to any pathological process.
- Never any fat reaction.
- Stains only with great difficulty.
- In above-mentioned localities is found only in man.
- Foci are larger, only irregularly spherical, and darker.

Besides the black pigment, which the author maintains is identical with melanin, yellow pigment and also, though far rarer, Marinesco's erythrophilic, paranuclear pigment were found in the cells of the locus cœruleus. The neurofibrils pierce the melanin masses, instead of encircling them as they do the pigment masses in senility or in amaurotic idiocy. The author regards all these pigments as being physiological metabolic products of past cellular activity.

ERNEST JONES.

PHYSIOLOGY.

THE CO-ORDINATION OF SINGLE MUSCULAR MOVEMENTS (612) IN THE CENTRAL NERVOUS SYSTEM. C. E. BEEVOR,

Journ. Amer. Med. Assoc., July 1908.

THE co-ordination of single muscular movements is, in this paper, considered under four headings: (1) prime movers; (2) synergic muscles; (3) fixation muscles; (4) antagonists. The writer points out that in every movement the prime movers contract in a regular orderly sequence, and that the muscles successively come into action, adding to their number according to the amount of work that is required to be done. Relation between the prime movers and the synergic muscles is very close, and it seems impossible to contract one without the other. Moreover, if a prime mover be weakened by a peripheral neuritis, the brain has no power to moderate the impulse sent to the synergics to make them act proportionately to the weak prime movers. It is pointed out that if a prime mover be paralysed, the patient has no power to contract a muscle which is in the secondary position of a fixation muscle, although he can do so when that same muscle is used as a prime mover.

The relaxation which takes place in an antagonistic muscle during the performance of a movement may be, as suggested by the author, of great practical use in two ways: (1) When difficulty is experienced in getting a patient to relax a given muscle, a sub-jacent joint or other structure can be easily examined by getting

the patient to use the muscle as an antagonist, when it will be relaxed ; (2) relaxation of the antagonists, which should normally occur, does not do so in many cases of functional paralysis, and the author considers this to be a symptom of functional disease as opposed to organic.

As to where, in the central nervous system, all these co-ordinations of single muscular movements take place, the author considers that all the evidence is in favour of the linkage of the ultimate constituents of a movement being in the spinal cord.

W. KELMAN MACDONALD.

STUDIES ON SENSORY CONDUCTION IN THE SPINAL CORD (613) BASED ON CLINICAL AND PATHOLOGICAL CONSIDERATIONS. (Studien über die sensible Leitung im menschlichen Rückenmark auf Grund klinischer und pathologisch-anatomischer Tatsachen.) H. FABRITIUS, *Sonderabdruck. a. d. Path. Instit. d. Univ. Helsingfors*, Bd. 2, Hft. 1. Karger, Berlin, 1907.

THIS study is based on two cases of cord injury (one with sectio) and the analysis of a large number of cases collected from various sources, all of which are given in tabular form. A translation is here given of the conclusions arrived at by the author:—

1. Mechanical stimulation of the skin originates a conduction stream which travels by two distinct paths in the spinal cord. Our sensations of touch and pressure arise through the simultaneous action of these two components on the brain cells.

2. One of these components is represented by conduction through the posterior columns, the other through the contra-lateral path.

3. Both touch and pressure sensations are conveyed by the first path, but the resulting sensation is lacking in tone.

4. This arises through the agency of the second component, which, indeed, calls forth the whole scale of sensibility from the faintest touch to the most severe pain.

5. Under certain conditions this component may become abnormally active relatively to the stimulation applied, and the tone of the resulting sensation will be unusually strong (hyper-æsthesia).

6. This hyper-activity probably arises through an increased reaction on the part of those posterior horn cells from which the tone-carrying contra-lateral paths arise.

7. The causes of this alteration are various. Sometimes it is a general toxæmia, or more frequently transverse lesions (injuries, hæmatomyelia, etc.), by which the fragile posterior horn cells are

suddenly torn out of their original relationships in the nervous system, especially their relationships with higher parts.

8. This isolation arises principally from lesions in the region of the cross pyramidal tracts. J. H. HARVEY PIRIE.

THE RATE OF TRANSMISSION IN HUMAN MEDULLATED
(614) **NERVE.** (Über die Leitungsgeschwindigkeit in den markhaltigen menschlichen Nerven.) PIPER, *Pflüger's Archiv*, Bd. 124, 11 and 12 Heft, 1908.

ATTEMPTS to measure the rate of transmission in human nerves have been made in various ways.

Helmholz measured the difference between the reaction times when stimulation is applied to points on the skin near to and distant from the central nervous system. By this method the rate of transmission in sensory nerves was found to be 60 metres per second, but the reaction time is very variable and the method uncertain.

More exact measurements are made by recording the contraction of the thumb muscles when stimulation is applied over the median nerve at two points successively. The rate has been found to alter with the temperature. Figures ranging from 17 to 87 metres have been given by observers who have recorded the contraction of muscles.

In the present research, the difference between the latent periods of the electrical reaction of the flexor muscles of the forearm was measured on stimulation of the median nerve by break induction shocks from the cathode of an induction coil.

The recording instrument employed was the small model of Einthoven's string galvanometer constructed by Edelmann.

The median nerve was stimulated at two points: (1) 5 cm. above the internal condyle of the humerus, and (2) in the axilla. The two points were distant about 16 to 17 cm.

The string of the galvanometer is affected by the stimulating shock through current escape or induction, and the author makes use of this as indicating the moment of stimulation.

The latent period of the muscle reaction to stimulation from the near electrode is, on an average, 0.00442 seconds; from the far electrode, 0.00578 seconds. The rate of transmission in the human median nerve, calculated from these figures, is 117 to 125 metres per second.

The measurements are made upon the assumption, founded on Einthoven's work, that his galvanometer reacts instantaneously.

The muscle contraction and action current are found to be greater when the nerve is stimulated above the condyle than when

the electrode is applied in the axilla. This is attributed not to variations in excitability in different parts of the nerve, but to the greater thickness of skin and adipose tissue in the latter position.

It is assumed that the latent period of the reaction does not vary with alterations in the strength of the stimulating current, and that excitability and conductivity are the same in different parts of one nerve.

It has been stated that temperature has an influence upon the rate of nerve transmission. The experiments recorded were performed in the month of June, and there remains the possibility that slower rates would be obtained in cold weather.

W. A. JOLLY.

ACTION OF BARIUM CHLORIDE ON THE FOWL'S MUSCLE.

(615) C. W. EDMUNDS and G. B. ROTH, *Amer. Journ. Physiol.*, Vol. xxiii., No. 1, Oct. 1, 1908, p. 46.

IN this short article barium is shown to act on the contractile substance of muscle cells, while curara, nicotine, and physostigmine act probably on some other constituent of the cell, which might be the "receptive substance."

Langley has postulated two constituents of cells:—

- (1) Substance concerned in the carrying out of the chief functions of the cells—secretion, contraction, etc.
- (2) "Receptive substance," capable of setting the chief substance into action, and which does not degenerate when the nerve is cut.

W. KELMAN MACDONALD.

PATHOLOGY.

CHANGES IN THE NERVOUS SYSTEM AFTER STOVAIN

(616) **ANÆSTHESIA.** (*Veränderungen des Nervensystems nach Stovainanästhesie.*) W. SPIELMEYER (of Freiburg), *Münch. med. Wochenschr.*, Aug. 4, 1908.

THE author examined the nervous system of thirteen cases who had been operated on under stovaine anæsthesia. In only one case was the stovaine the cause of death. In six cases .05-.07 grammes of stovaine were administered; in seven cases .12 or .1 gr. In nine of the cases examined, including the whole of the first group, no characteristic pathological changes were observed. The cell changes were such as to be most naturally explained on the basis of the general condition of the patient. In the case where the injection led to paralysis of respiration the cell changes were

apparently due to the respiratory disorder. In three cases characteristic cell changes were found; the alteration was of the nature of axonal reaction. The cells affected were the anterior cornual cells of the spinal cord. No changes could be demonstrated in the posterior roots or peripheral nerves, but they might easily have escaped observation as comparatively few cells were affected.

Experimental work on dogs and apes produced the same cell changes, and in addition changes in the posterior roots and also in the posterior columns. In one case the periphery of the lateral and anterior columns was also affected. The author calls attention to the fact that the cell changes do not necessarily lead to the destruction of the cells, and that damage of a few motor cells may produce no clinical evidence of weakness in the general musculature, but may be sufficient in the case of the eye nuclei to cause transitory paresis.

C: MACFIE CAMPBELL.

**A CONTRIBUTION TO THE PATHOLOGICAL ANATOMY OF
(617) MULTIPLE SCLEROSIS, WITH PARTICULAR REFER-
ENCE TO THE AREAS OF DISEASE MET WITH IN THE
CORTEX. (Zur pathologischen Anatomie der multiplen
Sklerose mit besonderer Berücksichtigung der Hirnrindenherde.)
GUSTAV OPPENHEIM, *Neurol. Centralbl.*, Okt. 1, 1908, S. 898.**

SUCCESSIVE sections of various parts of the brain and cord from four cases of disseminated sclerosis were stained by different elective tissue methods with the object of obtaining as complete a picture as possible of the alterations in the component tissue elements. Disappearance of the white sheaths and integrity of the fibrillar structure of the nerve elements in the islets of disease were demonstrated by the nerve sheath method of Weigert and the method of Bielschowsky respectively. In Marchi preparations some islets remained unstained, while in others considerable quantities of fat were found, chiefly at the periphery of the patches, often indeed forming a complete ring around the central apparently older portion of the patch. The nerve cells, axis cylinders, and fibrils were relatively intact. A special study was made of patches which involved both the cortex and subjacent white matter. Whereas the subcortical portion of these patches presented a more or less dense feltwork of neuroglial fibres, in the cortex itself the neuroglial increase consisted of an increase of spider cells. This accounts for the statements of observers, who worked before the introduction of the method of Weigert, to the effect that subcortical patches did not extend into the cortex, but terminated sharply at the junction of

the grey and white matter. In three of the four cases examined there was a widespread adventitial plasma cell infiltration, an appearance in favour of the exogenous cause of the disease. The absence of inflammatory changes in the fourth case does not, in the author's opinion, exclude this possibility, for it may be that the appearances met with were merely the later features of a process which in its earlier stages was of an inflammatory nature.

EDWIN BRAMWELL.

THE PRIMARY LESIONS OF THE NERVE FIBRES IN (618) URÆMIA, STUDIED IN EXPERIMENTAL CONDITIONS BY THE METHOD OF DONAGGIO. (*Le lesioni primarie delle fibre nervose nell'urinaemia and studiate in condizioni sperimentali con la colorazione positiva di Donaggio.*) SCARPINI, *Riv. di Patol. nerv. e ment.*, Agosto 1908, p. 349.

THE author uses the method of Donaggio, which may be thus briefly described. Pieces of tissue are fixed in a 4 per cent. solution of bichromate or in Muller's fluid. They are stained by hæmatoxylin and then subjected to mordant action of three types, *i.e.* the double chloride of tin and ammonia is used simultaneously with the staining (prima modalità of Donaggio), or the acetate of copper or perchloride of iron used subsequently to staining (second and third modalità).

The tissue is then subjected to decoloration by the method of Pal, which is carried on further than the stage of differentiation.

After such treatment fibres in which primary or secondary degeneration is commencing resist the action of the permanganate and oxalic and remain intensely stained among the normal fibres, which are colourless. Tissues which have been a very long time in chromic acid solution give this reaction.

The two first of these three reactions of Donaggio give the following results:—

The first renders visible the most minute structural differences in the altered fibre. The second, by giving the fibre a uniform coloration, allows it to be isolated with precision from the normal fibres.

The paper is a preliminary communication of the changes found in three rabbits dying after ligature of the ureters. In the spinal cord a certain number of fibres belonging to the tracts of Gowers and Flechsig were found to be affected. In two of the rabbits there were also lesions in the posterior columns, which were completely absent in the third.

In the bulb the restiform bodies showed numerous affected fibres, and a few of the internal arcuate fibres were affected.

Beyond referring to the singular symmetry of the lesions, the author makes no remarks on his pathological findings, but presents the paper as a preliminary note, showing the possibility of localising the nervous lesions in toxic affections.

F. GOLLA.

CLINICAL NEUROLOGY.

NERVOUSNESS AND NUTRITION IN CHILDHOOD. (*Nervosität (619) und Ernährung im Kindesalter.*) SIEGERT, *Munch. med. Woch.*, Sept. 22, 1908, p. 1963.

IN this most interesting paper the "nervous" child discussed is the child that shows symptoms of nervousness owing to unsatisfactory feeding.

The condition is frequently brought about by overfeeding, especially with albuminous substances. Excess of meat, eggs, and milk is the common cause, and the results are most marked when at the same time there is lack of green vegetables and fresh fruit in the diet.

The author proceeds to describe some typical cases of the condition in which the nervousness is only one symptom—there are many others.

Cure is only brought about by regulation of the diet—by cutting down the amount of albuminous food, and often also the fat, and by increasing the amount of vegetables and fruit. The condition is exceedingly common.

A. DINGWALL-FORDYCE

REPORT OF A CASE OF MYASTHENIA GRAVIS PSEUDO-
(620) **PARALYTICA, WITH NEGATIVE PATHOLOGICAL FINDINGS.** J. ARTHUR BOOTH, *Journ. of Nerv. and Ment. Dis.*, Nov. 1908, p. 690.

THIS is a record of a case of myasthenia gravis occurring in a boy aged 11. The symptoms, which came on gradually a few days after an operation for adenoids, presented nothing exceptional. Death occurred eighteen months later. Post-mortem a slight enlargement of the thymus gland was found. Otherwise nothing abnormal was detected. The muscles, central nervous system, thyroid, parathyroids, and pituitary body were all carefully examined. In view of the negative findings in this and similar cases "the symptom complex of the disease is best explained on the basis of its being a nutritional disorder impairing the vital processes in the muscles from some unknown toxine."

EDWIN BRAMWELL.

**OCCUPATION NEURITIS OF THE DEEP PALMAR BRANCH
(621) OF THE ULNAR NERVE.** J. RAMSAY HUNT, *Journ. of
Nerv. and Ment. Dis.*, Nov. 1908, p. 673.

THE author describes three cases of what he regards as an occupation neuritis characterised by an atrophic paralysis of all the intrinsic muscles supplied by the ulnar nerve, the reaction of degeneration in the atrophic muscles, and absence of any objective sensory disturbance in the ulnar nerve distribution. Sensation is unaffected, therefore the nerve must be involved after the superficial or sensory branch has left the motor, while the fact that all the muscles supplied in the hand by the ulnar nerve are affected shows that the nerve has been implicated before it breaks up to supply the individual muscles.

The paralysis is caused, he believes, by pinching of the nerve as it passes between the abductor and short flexor muscles of the hypothenar eminence near their origin.

The occupations of the three patients referred to were those of jeweller, a machinist, and a brass polisher. The cases recorded by Gessler, in the opinion of the author, in all probability belong to the condition here described.

EDWIN BRAMWELL.

ACUTE POLIOMYELITIS FOLLOWING TONSILLITIS. KENDALL
(622) EMERSON, *Boston Med. and Surg. Journ.*, 1908, p. 500.

A BOY, aged 8 years, three days after recovery from a severe attack of tonsillitis, in which no diphtheria bacilli were found, complained of tingling in the left hand. Two days later loss of the left triceps reflex and weakness of the left arm muscles occurred. During the following five days the development of the paralysis was progressive until all power was lost except weak flexion of the fingers. Emerson regards the tonsils as the atrium of infection for the toxins, which later affected the cord, and considers that the bodily resistance was lowered by the boy refusing to rest after the initial attack.

J. D. ROLLESTON.

CASE OF CHRONIC ANTERIOR POLIOMYELITIS. (Über einen
(623) Fall von Poliomyelitis anterior chronica.) R. CASSIRER and
OTTO MAAS, *Monatsschr. f. Psychiatr. u. Neur.*, Oct. 1908,
Bd. 24, S. 306.

THE following case is fully and carefully described. A woman of sixty suffered from weakness of the legs, which gradually ascended and affected the trunk and arms. Marked atrophy was present in the legs. The paralysis was throughout a flaccid one. She died in three years, and at the autopsy was found extensive degeneration

of the anterior horn cells, with, as is the rule in such cases, a few scattered lesions in the white substance. The pathological appearance is especially fully described.

The diagnosis lay between progressive muscular atrophy and chronic anterior poliomyelitis. Pointing to the latter was: the continuous spread of the affection, no special groups of muscles being picked out, the onset in the legs, the chronic course during which the neck and bulbar muscles were quite unaffected.

ERNEST JONES.

ON THE QUESTION OF IDIOPATHIC "MENINGITIS SPINALIS (624) SEROSA CIRCUMSCRIPTA." (Zur Frage der idiopathischer Form der "meningitis spinalis serosa circumscripta." I. BRUNS, *Berl. klin. Woch.*, Sept. 28, 1908, S. 1753.

IN view of the fact that doubt has been cast on the existence of an "idiopathic" form of this (unquestionably rare) condition even by those who have described cases, the author reports here at greater length a case previously briefly recorded by him. The case was that of a boy of sixteen with all the symptoms well marked of an extra-medullary tumour pressing on the cord about the level of the 4-5 cervical segments. At the operation there was found only a circumscribed accumulation of cerebro-spinal fluid under high tension. There was nothing to which this congestion could be said to be secondary. Removal of the fluid resulted in almost complete recovery from the pressure symptoms. When seen twenty months later there was no relapse and practically no sign of the disease remaining. The author is strongly of the opinion that such an idiopathic condition exists apart from any disease of the spine, membranes, or cord itself.

J. H. HARVEY PIRIE.

TWO CASES OF LANDRY'S PARALYSIS. (Zwei Fälle von (625) Landry'scher Paralyse.) ARTH. V. SARBO, *Neurol. Centralbl.*, Nov. 1, 1908, p. 1009.

TWO cases are here described as Landry's paralysis. The first terminated fatally after a four days' illness, while the second recovered.

CASE 1.—A boy, aged 12, in the best of health, was suddenly affected with paralysis of the palate and difficulty in swallowing. On the second day the weakness was more pronounced, and there was paralysis of the right side of the face, which was followed quickly by left facial and right sixth paralysis, while on the same day weakness of the neck muscles, upper extremities, and thorax were observed. On the fourth day the left knee-jerk disappeared

and the tendon-jerks on the right side were more difficult to elicit than previously. Death occurred on the fourth day with paralysis of the intercostals and diaphragm. The only point in the previous health was a discharge from the one ear, which dated back for five years. The author lays stress on the absence of sensory symptoms, and regards the case as a descending Landry's paralysis. He refers to several similar cases in the literature. No autopsy was made.

CASE 2.—A woman, aged 19, who had married against the will of her father, came home after a three weeks' wedding tour complaining of subjective sensations and weakness in the feet, which symptoms were followed by complete paralysis after a stormy interview with her father. The patella and Achilles reflexes were found to be absent. She complained of severe pain in the dorsal region of the spine. Paralysis of the trunk, the arms, the face, and the soft palate quickly followed, with hyperæsthesia of the extremities and the picture of an ascending motor paralysis. Improvement began to occur on the tenth day. There was, however, muscular atrophy, with reaction of degeneration, but no sensory disturbances or fibrillary tremors. After fourteen weeks she was almost well, although the knee-jerks were still absent. No organisms were found in the blood. EDWIN BRAMWELL.

TRAUMATIC AFFECTIONS OF THE CORD. (Zur Kenntniss der (626) traumatischen Rückenmarksaffectiionen.) WINKLER und JOCHMANN, *D. Zeitsch. f. Nervenheilk.*, 1908, Bd. 35, S. 222.

THESE authors give a detailed clinical and anatomical account of two cases of hæmatomyelia. The first was in a man of forty-five, who was struck on the back by a railway signal bar four months before admission. This produced an extensive intramedullary hæmorrhage in the cervical and upper thoracic portions of the cord. The second was in a school girl of twelve, who fell a distance of about two metres from a ladder during gymnastic exercises. No serious symptoms developed until the following day, when pain and weakness appeared in the left arm and leg. The patient survived eighteen months, and died ultimately from bedsores. The hæmatoma was chiefly in the 3rd, 4th, and 5th cervical segments.

The details of these cases are carefully set forth and well illustrated by diagrams, but cannot well be condensed in an abstract. PURVES STEWART.

TRAUMATIC CERVICAL MYELOMALACIA. CARL D. CAMP, (627) *Journ. Amer. Med. Assoc.*, Aug. 22, 1908.

THE case which is reported in this paper is that of a man who fell about six feet, striking his back on the frozen ground. After

the accident there was a prominence in the cervical region of the spinal column, but next day there was no deformity of the spine in any part, and radiograph showed nothing abnormal. The paralysis was peculiar, inasmuch as it apparently advanced and receded and was of a peculiar distribution for an organic lesion of the cord; both forearms and one leg seemed to be the seat of permanent paralysis, while the paralysis of the other leg apparently appeared one day to disappear the next. There was no Babinski reflex, but there was a band on the chest of true *allochiria* and other various and varying disturbances of sensibility. A month later atrophy of the paralysed parts set in.

The patient died four months after the accident, and the autopsy revealed a linear fracture in the body of the sixth cervical vertebra, with an area of softening in both anterior horns from the sixth cervical down to the first thoracic segment. There was no evidence of an old hæmorrhage, and the author explains the condition as being due to a slight dislocation of the vertebræ pressing on the spinal cord sufficiently to block the anterior spinal artery, setting up a thrombosis which caused areas of softening in the anterior horns and to some extent the adjacent white matter.

The diagnosis from traumatic hysteria is discussed, and the inadvisability of surgical interference is emphasized.

W. KELMAN MACDONALD.

THE CEREBRO-SPINAL FLUID IN DIPHTHERITIC PSEUDO-TABES. (628) **TABES.** (*Zur Klinik postdiphtherischer Lähmungen. Liquorbefunde bei postdiphtherischer Pseudo-tabes.*) RÖMHELD, *Neurol. Centralbl.*, 1908, p. 1007.

At the annual meeting of German neurologists at Heidelberg, Römhelt recorded the first case of diphtheritic paralysis, in which an examination of the cerebro-spinal fluid has been published. The patient was an adult who had been treated with antitoxin. After suffering from paralysis of the palate and accommodation, three months after the initial attack he presented marked ataxia of the upper and lower limbs, loss of tendon jerks, tenderness of the nerve trunks, diminution of electrical excitability and subjective sensory disturbances. The first lumbar puncture was performed two and a half months after the onset of diphtheria. Moderate lymphocytosis was found. A month later there was hardly any increase in the lymphocytosis, and the amount of albumin in the fluid was less. No diphtheria toxin nor antitoxin was present. At the third puncture, performed a month later, the cellular contents were normal and the albumin was still less. On his discharge the patient was in a normal condition, except

that his tendo Achillis jerks were absent and his knee jerks were weak. Since no pathological change is present in ordinary peripheral neuritis, these observations show that in diphtheritic paralysis of long duration there exists not only peripheral neuritis but central anatomical changes. A comparison is made with the findings in the cerebro-spinal fluid in metasymphilitic processes. Whereas in the latter the morbid changes once present persist, in diphtheritic paralysis the diminution in the cellular contents and amount of albumin goes hand in hand with clinical improvement.

J. D. ROLLESTON.

CYTOLOGICAL EXAMINATION OF CEREBRO-SPINAL FLUID (629) AND ITS INTERPRETATION. (*Ergebnisse der zytologischen Untersuchungen der Zerebrospinalflüssigkeit und deren Ausichten.*) O. REHM, *Münch. med. Wochenschr.*, Aug. 1908, p. 1636.

DR REHM gives a short preliminary account of the history of lumbar puncture from its therapeutical and diagnostic aspects. Introduced therapeutically in 1891 by Quincke, its uses, from a diagnostic point of view, were quickly recognised, and the fluid was examined to determine the pressure, at which it was expelled, its chemical constitution, more particularly the presence or absence of albumin, the bacteriological examination of the fluid, the presence of cellular elements and their characters, whether lymphocytes or leucocytes, in the different pathological conditions examined. Valuable work was early done by various French workers in syphilitic, post-syphilitic, and many associated diseases of the central nervous system.

Nissl was the first in Germany to classify those diseases in which an increase in the number of lymphocytes is present, and he also confirmed many of the results of French writers. He emphasised the fact that both the exact number and the kind of cell present must be noted. The so-called French method then in use, consisted in centrifuging a definite amount of the fluid for a definite time, collecting the sediment in a hair-pipette, and blowing this out in drops on a slide, which are then fixed and stained. This method leaves too much room for variations in technique, slight errors in which will markedly affect the result, and it diminishes the value of comparison between the results of different observers. In 1904 Fuchs and Rosenthal used a counting chamber similar to that used in the enumeration of blood corpuscles. This is a more exact method than the former, and is of great value if the cells are numerous, and in comparing results in the same case from day to day. Alzheimer, in 1907, introduced a new method of fixing the cells. He saturated the

fluid with 96 per cent. alcohol, centrifuged the coagulum, hardened, fixed, and embedded it in celloidin; this he could then cut and stain. In cases where the albumen is distinctly increased this method is very valuable, but in others the coagulum is so thin as to render the process very difficult.

Dr Rehm then describes his results after a series of 650 punctures. He classifies them as follows:—From one to five cells in cubic millimetre is normal, from six to nine he puts in a sort of neutral zone between normal and pathological. In this group many of the cases had a syphilitic taint, besides some cases of fever delirium, and several cases of tabes and progressive paralysis. He begins the distinct pathological increase with ten cells per cubic millimetre. Some much higher counts he obtained in meningitis and meningomyelitis.

Dr Rehm found in syphilitic cases a distinct diminution in the number of cells after a course of mercurial inunction, thus showing that the mercury has a distinct influence on the cell production. He then goes on to describe the kind of cells found in various diseases, and gives a very full account of their appearance; he finds the Alzheimer method much the more suitable for exact differentiation of the cells.

Dr Rehm concludes the article by emphasising the great importance of lumbar puncture as part of the routine examination of patients and the careful examination of the kind of cells present, considering the results to be obtained as valuable as those which have been got in the blood examination in other cases.

DUNCAN LORIMER.

ACUTE ASEPTIC MENINGITIS. (*Contribution à l'étude des (630) réactions méningées aiguës aseptiques.*) L. CHABBERT, *Thèses de Paris*, 1907-1908, No. 159.

ACUTE aseptic meningitis occurs in syphilis of the nervous centres, otitis media, herpes zoster, and after injection into the pia-arachnoid cavity of stovaine or cocaine. The symptoms are the same as in the other varieties of meningitis. Diagnosis can only be made after lumbar puncture. The cerebro-spinal fluid in these cases has two principal characteristics—(1) It is aseptic, or, at least, the present laboratory methods show no micro-organisms; (2) it contains polymorphonuclears which are remarkable for their perfect state of preservation. Recovery is the rule, but certain symptoms may persist, such as headache, apathy, inequality of the pupils, and dissociation of the pulse and temperature. The treatment is the same as in septic meningitis, except that one should abstain from injecting substances into the pia-arachnoid cavity which might increase the intensity of the reaction.

J. D. ROLLESTON

TUBERCULOUS MENINGITIS IN PREGNANCY. (De la méningite (631) tuberculeuse au cours de la grossesse.) H. PLIVARD, *Thèses de Paris*, 1907-1908, No. 342.

TUBERCULOUS meningitis is exceptional during pregnancy. It may occur at any stage, unlike eclampsia, which is rare before the sixth month. It may assume either an epileptiform or a comatose character, and therefore closely resembles eclampsia, from which it is to be distinguished mainly by the character of the pulse. Examination of the urine is of little help, since albuminuria may be present in tuberculous meningitis. Since tuberculous meningitis usually forms part of a more or less generalised tuberculosis, search must be made for evidence of the disease in other organs, especially the lungs and choroid. Lumbar puncture and the ophthalmo-reaction will also be of assistance. The thesis contains the histories of seven cases, two of which have hitherto been unpublished. In three cases the disease was manifested by both convulsions and coma. In four coma was present, but the convulsions were ill-marked or absent.

J. D. ROLLESTON.

TREATMENT OF CEREBRO-SPINAL MENINGITIS WITH (632) FLEXNER'S SERUM. C. B. KER, *Edin. Med. Journ.*, Oct. 1908.

BEFORE the serum prepared by Flexner and Jobling was used, the death-rate of the cerebro-spinal cases at the Edinburgh City Hospital was not less than 80 per cent. At first the treatment adopted was chiefly expectant in character, but most of the serums obtainable were given a trial. In all, thirty-three patients were treated with Flexner's serum, the mortality being 42·3 per cent., or about half that of the cases treated otherwise. Several of the patients injected were moribund or quite hopeless on admission. If these are deducted the death-rate falls to 33 per cent. These figures, while perhaps not so satisfactory as those reported by other observers, are extremely encouraging.

The serum was invariably injected into the spinal canal, and an average adult dose was 30 c.c. As much spinal fluid as would readily escape was withdrawn before each injection. The best results were obtained by giving daily injections for the first three days the patient was under observation, and if improvement was not noted within that time the serum was pushed, doses being given at intervals, as long as the symptoms remained acute and meningococci were present in the spinal fluid. The largest amount

of serum given in any one case was 360 c.c. In favourable cases the injections were followed by improvement in the general condition of the patient. The rigidity and headache rapidly disappeared. Occasionally a fairly definite crisis occurred, a mode of termination which had not been observed in untreated cases. The spinal fluid rapidly became less turbid and the extra-cellular micro-organisms disappeared early, all the cocci becoming intra-cellular. The staining properties altered, the organisms appeared disintegrated, and after a few days' treatment it often became difficult to get cultures to grow. Ker does not lay any stress on the opsonic and agglutinative reactions as aids in prognosis, but believes that both are of considerable value in diagnosis.

A great feature in the cases, which ended in recovery, was the complete absence of chronic symptoms. Previously the patients who survived had for the most part a long and tedious illness, and frequently became quite deaf. Much wasting had also been observed. The patients, however, who were treated with Flexner's serum made rapid recoveries, and, with two exceptions, left hospital perfectly well. The serum was also most effective in checking the relapses so often met with in cerebro-spinal meningitis. The small mortality of children of under ten years of age, only 20 per cent., was another very striking feature. To obtain success it is very necessary to inject early, and, provided this is done, there is hope even for patients with pus in the spinal canal.

Ker also treated fourteen patients with Kolle's serum. Of these, eight died. The injections, however, in this series were almost entirely subcutaneous, and he concludes that, whatever the merits of this particular serum may be when used in large doses intraspinally, there is little to be gained by subcutaneous injections. On the other hand this serum is recommended as worthy of a trial in large intraspinal doses. Jochmann's serum was also tried and given much on the same lines as Flexner's, but with very poor results.

AUTHOR'S ABSTRACT.

**THE SERUM TREATMENT OF CEREBRO-SPINAL FEVER IN
(633) THE CITY OF GLASGOW FEVER HOSPITAL, BELVIDERE,
BETWEEN MAY 1906 AND MAY 1908. J. R. CURRIE and
A. S. M. MACGREGOR, *Lancet*, Oct. 10, 1908.**

WITHIN the periods quoted Belvidere Hospital received 330 cases of cerebro-spinal fever. Of these, 105 were treated with anti-meningococcic serum, while 225 were not so treated. The results of administration were checked throughout by control cases of a similar type, and the fallacy avoided of comparing the treated cases of one stage of the epidemic with the untreated cases of

another. The four sera employed were Professor Wassermann's, Professor Ruppel's, Professor Kolle's, and Messrs Burroughs, Wellcome & Co.'s. An account is given of the clinical features of the epidemic, and a comparison of the mortality rate with that obtaining in other and previous epidemics brings out the fact that the Glasgow type was one of great severity (mortality = 74.8 per cent.).

The authors' experience is discussed from (a) the clinical, (b) the statistical aspects. Clinically it was found that the administration of serum was followed in individuals by no consistent modification of the natural course of the disease. A tabular inquiry was conducted into (a) total case mortality among treated and untreated cases; (b) duration of illnesses ending in death; (c) duration of illnesses ending in recovery; (d) relative mortality among cases that survived the first ten days of illness. Briefly, the authors are unable to report that total case mortality was reduced, or that fatal issues were delayed, or favourable illnesses curtailed. But it was found that when serum-treated survivors of the first ten days were compared with untreated survivors in respect of case mortality, the treated cases recovered in greater number, sufficiently so to allow the inference that serum-treated cases which survived the first ten days had a better chance of life. This result suggests that treatment in these cases, though failing to arrest the disease out of hand, was yet able to hamper its progress, aiding the natural defences of the body and hindering, possibly, the formation of exudates which would stand in the way of recovery.

It is pointed out that treatment directed against the infective process is limited on the one hand by the extreme severity of the infection during epidemic periods, and on the other, in the later stages of the case, by the occurrence of gross pathological changes which prejudice recovery.

AUTHORS' ABSTRACT.

THE CORTICAL LOCALISATION OF ASYMBOLY. (*Die kortikale (634) Lokalisation der Asymbolie.*) E. POGGIO, *Neurol. Centralbl.*, Sept. 1, 1908, S. 817.

A CASE of Jacksonian epilepsy. Some of the attacks began with contractions in the last three fingers of the left hand, others with rotation of the head and eyes to the left. Two localised cortical lesions were diagnosed. Nothing objective was detected on examination, in particular there was no motor weakness and no astereognosis. An operation was performed, and two hydatid cysts about the size of a cherry-stone were found lying on the surface of the brain and removed. Twenty-two days after the

operation, apart from a very slight tactile loss in the tips of the fingers, asymboly in the left hand was the only sign of disease. Thus, when an object was placed in the patient's left hand, his eyes being closed, although he could describe accurately its form and properties, he was unable to say what it was. When the object was placed in the right hand he named it at once. The case is analogous to those reported in which an object cannot be named at sight although it is recognised when handled.

EDWIN BRAMWELL.

**ON THE SYMPTOMATOLOGY OF CEREBELLAR AND EXTRA-
(635) CEREBELLAR TUMOURS.** (Zur Symptomatologie der
Tumoren des Kleinhirns und des Kleinhirnbrückenwinkels.)
GIERLICH, *Deutsche Medizinische Wochenschrift*, No. 42, p. 1800,
Oct. 1908.

THE author gives a detailed account of two interesting cases.

CASE I.—The case of a boy, aged 7, suffering from a sarcoma growing from the inferior vermis of cerebellum into the fourth ventricle. The following is a brief account of the case and post-mortem findings.

The patient, aged 7, in whose previous history nothing noteworthy had occurred, in December 1906 had suffered for about two months with frontal headache, nausea, and giddiness. Vision was gradually becoming impaired. An acute attack of vomiting and giddiness brought him under medical supervision. Gait—drunken type, unsteadiness on standing, increased by closing the eyes. No muscular weakness. Tremor in arms and legs increased on voluntary movements. No true ataxy of arms. Increase of deep reflexes, ankle clonus, and extensor responses. Definite spasticity in legs. No sensory loss. Nystagmus on lateral deviation of the eyes. Double optic neuritis.

A diagnosis of cerebellar tumour, or possibly meningitis serosa, was made.

The patient grew steadily worse—all symptoms becoming more marked. In addition, definite ataxy and adiodocinesia were observed on the right side. An operation was performed, the whole cerebellum exposed and a tumour mass seen projecting into the posterior fissure of cerebellum. The patient died shortly after operation.

Post-mortem.—Great hydrocephalus internus. Frontal section through the cerebellum showed a tumour growing from the inferior vermis, and projecting into the fourth ventricle. It was easily removed from its surroundings, and proved to be a round-celled sarcoma. The author points out that extreme ataxy is often met with in lesions of the inferior vermis, and especially, as in this

case, of its posterior part. A lesion in this position will interfere with fibres from the cerebellum to Deiter's nucleus, and also with the spino-cerebellar tracts.

CASE II.—Extra cerebellar tumour(neurofibroma)growing from the ninth cranial nerve.

The time from onset of symptoms to the patient's death was four years. The first symptoms noted of any localising value were loss of smell and primary optic atrophy. Subsequently unsteadiness in walking developed, with loss of taste on right side of the tongue and evidence of implication of the right fifth, seventh, and eighth nerves. The palate moved very little, and the palate reflex was lost. Late in the disease difficulty in swallowing fluids and solids developed, and the patient died during a tonic convulsion, which at the end were of almost daily occurrence. The interest of the case lies in the fact that the first symptoms of localising value were not, as was supposed, due to a growth in the anterior fossa, but were simply evidence of hydrocephalus internus caused by an extra-cerebellar tumour growing from the ninth cranial nerve. The cerebellar symptoms and cranial nerve palsies were supposed to be due to a second tumour, and on this account no operative interference was undertaken.

C. M. HINDS HOWELL.

THROMBOSIS OF SUPERIOR LONGITUDINAL AND LATERAL (636) SINUSES, COMPLICATED BY PREGNANCY. TREATED BY OPENING THE TORCULAR HEROPHILI. DEANE, *Journ. Amer. Med. Assoc.*, Sept. 19, 1908, p. 997.

THE author believes this to be the first case of sinus thrombosis on record treated by opening the torcular Herophili. The patient was a married woman, aged 18 years, who for eleven years previously had had a discharge from the right ear. She was admitted to hospital with urgent symptoms. Operation revealed mastoid suppuration and gangrene of the wall of the lateral sinus, with absence of bleeding on incision. Probing downwards resulted in some hæmorrhage, probing upwards produced hardly any. Five days later the internal jugular was ligatured; and after three days more the jugular was excised. Cultural examination of the blood revealed the presence of the staphylococcus pyogenes aureus. Severe symptoms of septicæmia still continued. The absence of eye symptoms seemed to exclude cavernous sinus thrombosis, and there were no signs of cerebral or epidural abscess. Sixteen days after the first operation the cerebellum was explored without any abscess being discovered. The right lateral sinus and the torcular Herophili were exposed and incised, no bleeding resulting. Septic material was removed with a curette from the superior longitudinal sinus, and curetting of the left lateral sinus was followed

by free hæmorrhage. The thrombosis had clearly affected the entire right lateral, and parts of the superior longitudinal, torcular Herophili, straight, occipital, and inferior petrosal sinuses, not less than fourteen inches of venous channel being obliterated. After the fourth operation the patient made a good recovery. The radical mastoid operation was completed later on. The patient was five months pregnant at the onset of the illness, but the course of gestation was not interrupted, and at full term she gave birth to a healthy infant.

HENRY J. DUNBAR.

THE CAUSES AND SYMPTOMS OF THROMBOSIS OF THE
(637) **CAVERNOUS SINUS.** ST CLAIR THOMSON, *Ophthalmic Review*,
Oct. 1908, p. 293.

THE author contends that disease of the sphenoidal sinus is the most common cause of thrombosis of the cavernous sinus. He has elsewhere reported sixteen cases collected from the literature, with post-mortem confirmation. The present short paper contains a number of diagrams illustrating the intimate anatomical relationship which exists between the cavernous sinus and the accessory sinuses of the nose.

EDWIN BRAMWELL.

CERTAIN ANEURYSMS OF CEREBRAL VESSELS. JOHN ROSE
(638) BRADFORD, *Lancet*, Sept. 5, 1908.

AFTER commenting upon the comparative frequency of aneurysm of the cerebral vessels, the author classifies the cases into four groups. In the first group of cases there are no symptoms, and the aneurysm is found accidentally at the autopsy when the patient dies of something else. The second group is made up of those which are merely cases of cerebral hæmorrhage, while cases which produce symptoms of cerebral tumour fall into the third group. The fourth is very important, and consists of a very definite group of cases characterised by these phenomena: (1) That there is an early seizure, followed by (2) a period in which there are no marked symptoms of any kind, perhaps with the exception of some stiffness of the neck; and (3) subsequently there is a fatal seizure. The opinion is expressed that the first seizure is due to a slight rupture and the fatal seizure is due to a second rupture, and great emphasis is laid upon the practical importance of clinically recognising the true prognostic significance of the first seizure.

W. KELMAN MACDONALD.

CEREBRAL ABSCESS. EMIL AMBERG, *Journ. Amer. Med. Assoc.*,
(639) Aug. 22, 1908.

THIS is an account of a case of a girl of 12 years who was operated on on the day of admission to hospital for middle ear disease, and a

thrombus of the lateral sinus removed. Nine days later the middle cranial fossa was opened and two drachms of yellowish-green pus evacuated. One week later the patient died, and at the autopsy, in addition to a chronic suppurative tympano-mastoiditis, an abscess in the region of the dura of the roof of the antrum and an extended lateral sinus thrombosis, an old deep-seated brain abscess was found in the left cerebrum adjacent to the ventricle. Examination of pus smears showed the diplococcus pneumoniæ and the colon bacillus.

W. KELMAN MACDONALD.

A CONTRIBUTION TO THE ETIOLOGY OF EPILEPSY. (Zur (640) *Ätiologie der Epilepsie*.) BRATZ, *Neurol. Centralbl.*, Nov. 16, 1908, S. 1063.

STATISTICAL observations by Sichel indicate that although many neuroses are more common among the Jewish race than among the general population, the reverse holds good for alcoholism and epilepsy. The author brings forward further statistics, and raises the question, is the rarity of epilepsy related to the infrequency of alcoholism? He has observed 1262 cases of idiopathic epilepsy at the Anstalt Wuhlgarten, of which 28 (2½ per cent.) were Jews (the Hebrew population of Berlin is estimated at 10 per cent. of the whole). Since, however, the Jews of Berlin are generally supposed to live in better circumstances than the general population of that city, there is a possible fallacy in connection with figures drawn from the material of a public institution. No instance of parental alcoholism was present among the 28 cases of epilepsy above referred to, although in every case there was a neuropathic taint. On the other hand, among the remaining 1234 cases a neuropathic heredity was present in 391, and a history of alcoholism in the parents in 254. Although some authorities assert that alcoholism in the parents acts as a special predisposing cause of epilepsy, it has always to be remembered that in almost all cases of alcoholism there is a neuropathic taint, and the epilepsy in the offspring may be merely the expression of a neuropathic heredity rather than a direct consequence of the toxic effect of alcohol upon the unborn child.

EDWIN BRAMWELL.

THE BLOOD IN EXOPHTHALMIC GOITRE AND IN THYROIDISM. (641) ISM. (*Blutbefunde bei Morbus Basedowii und bei Thyreoidismus*.) L. CARO, *Berl. klin. Wochenschr.*, No. 39, 1908.

THE writer had full blood examinations carried out in fourteen cases of exophthalmic goitre, in twenty cases of thyroidism without

thyroid enlargement, in six cases of goitre without symptoms of thyroidism, and in eight normal cases after the administration of thyroid tablets. He comes to the following conclusions:—

(1) Well-marked cases of exophthalmic goitre show a striking reduction of the polymorph leucocytes (to 50 per cent.), along with a marked increase of lymphocytes (up to 50 per cent.), the increase being chiefly in the small lymphocytes.

(2) Relative lymphocytosis is found in the fruste forms of exophthalmic goitre, *i.e.* cases of thyroidism.

(3) In cases of simple thyroidism the large mononuclear cells are increased more than are the small lymphocytes, a relative increase of the latter being typical of a fully developed case of exophthalmic goitre.

(4) In cases where clinically the exophthalmic goitre appears to be cured, the blood picture approaches the normal standard, but a slight relative increase in the large mononuclear cells is often found in such cases, and indicates that a slight degree of thyroidism is still present.

(5) In normal cases fed with thyroid tablets a relative lymphocytosis is invariably found, the large mononuclears being chiefly involved.

The writer considers that in cases of cardiac disturbance the presence of a lymphocytosis is strong evidence in favour of thyroid intoxication being the cause.

D. P. D. WILKIE.

ON THE PSYCHICAL NATURE OF BLEPHAROSPASM. (Sulla (642) *natura psicogena del blefarospasmo.*) GEROLAMO MIRTE, *Ann. di Neurol.*, An. 26, Fasc. 1, 1908.

THE author quotes the definition of Bianchi of tic and epilepsy as being both "motor disorders of irrational and inefficient type which are propagated by areas of cortex not under control of the general laws of co-ordination, of subordination, and of association."

Encouraged by the results published by Dr Valude of Paris, he attempted the treatment of two of his cases by injection of alcohol into the seventh nerve at its point of emergence. In each case the injection was followed by paralysis of the facial muscles, but as the paralysis passed off after a duration of three to four months, the blepharospasm returned and regained its former intensity.

In one case after failure of the injection treatment, and as the patient had persistent photophobia, the ophthalmic division of the fifth was resected on one side, but without any effect. Treatment by galvanism proved useless. Instillation of atropine relieves the symptoms for a few hours, but has no curative effect.

The author then proceeds to discuss the pathogenesis of blepharospasm. He considers that an organic excitatory cause can only be found in a small number of cases. In one of his cases photophobia might be looked upon as an excitatory cause, but inasmuch as section of the trigeminal failed to relieve it, this view cannot be maintained.

The author concludes with Oppenheim that facial spasm is a psychogenic phenomenon that constantly repeats its original cause; that is, a strong emotion or the continual mental disturbance to be found in such cases, and that the spasm chiefly affects individuals with a strong hereditary neuropathic tendency.

He considers that the symptoms of blepharospasm afford no support to the attempt of Brissaud to differentiate spasm as a purely subcortical reflex phenomenon from tic as an automatic cortical discharge.

F. GOLLA.

THE ATTENTION NEUROSIS. (*Die Erwartungsneurose.*) M. (643) ISSELIN (of Munich), *Münch. med. Wochenschr.*, July 7, 1908.

THE neurosis of attention or expectation discussed by the author consists in the disturbance of various more or less automatic activities by the anxious direction of the attention to their execution. Instead of normal functioning there is hesitation, and defective action even to the extent of paralysis, the individual act being preceded by pronounced distress and a feeling of tension. The neurosis usually remains limited to one special form of activity, *e.g.* writing, reading, sleeping (insomnia), speaking, swallowing, micturition ("stuttering micturition"), walking (astasia-abasia), etc. The neurosis usually develops on the basis of a psychopathic constitution, with a natural tendency to indecision and worry. The function affected is usually determined by some actual difficulty or disorder. The disorder arises when for some reason or another the attention of the individual is specially directed to an otherwise automatic activity; thus a "stuttering" gait may ensue after prolonged rest in bed. Inability to write may be caused by temporary over-fatigue; inability to swallow easily may result from the attention paid to the act during a tonsillitis. The patients do not present the hysterical constitution; the symptoms remain of very limited range in each individual case.

There is only one satisfactory mode of treatment, namely, psychotherapy, and especially hypnosis.

C. MACFIE CAMPBELL.

POSTSCARLATINAL CONVULSIVE URÆMIA. (*Considérations (644) sur un cas d'urémie eclamptique postscarlatineuse.*) P. NOBÉCOURT and P. HARVIER, *Bull. et mém. de la Soc. méd. des Hôp. de Paris*, 1908, p. 383; LÉON BERNARD, *ibid.*, p. 409.

A BOY, aged 12 years, had a slight sore throat, not followed by an eruption. A month later, while in apparently good health, he was seized with epileptiform convulsions. The urine was scanty and smoky, and contained a large quantity of albumin. The diagnosis was made of convulsive uræmia in the course of acute nephritis, probably of scarlatinal origin. The convulsions disappeared on the fourth day, and complete recovery took place. During the attacks there was exaggeration of the left knee-jerk and Babinski's sign on the same side. The attacks were followed by stiffness of the neck, Kernig's sign, and complete abolition of both knee-jerks for a month. Kernig's sign was not due to a meningeal reaction, since no cells were present in the cerebro-spinal fluid, but was probably the result of uræmic intoxication of the cerebral cells. The writers do not attempt to explain the temporary abolition of the knee-jerks. At the subsequent meeting Bernard stated that the existence of scarlet fever was by no means proved in this case. There was too great a tendency, he thought, especially among pædiatrists, to attribute every acute nephritis of obscure origin to scarlet fever. Acute nephritis following tonsillitis had been shown to exist by Bouchard and Landouzy, and Bernard himself had seen such cases.

J. D. ROLLESTON.

TREATMENT.

SOMETHING ABOUT PUNCTURE OF THE BRAIN. (Brit. (645) Med. Assoc.) TILLMANNS, *Brit. Med. Journ.*, Oct. 3, 1908, p. 983.

PUNCTURE of the brain has not received the attention that it deserves, and, like lumbar puncture, it will certainly come to be more and more universally practised both as a diagnostic and as a therapeutic measure. Diagnostic puncture is indicated when there are signs of cerebral pressure, where abscess, hæmorrhage, and the like are suspected, and for the localisation of cysts, tumours, and foreign bodies. Therapeutic puncture has been principally made use of in hydrocephalus and for the injection of tetanus antitoxin. In performing the puncture the head is prepared as for trephining, local or general anæsthesia is employed according to

circumstances, and a brace and drill are used either with or without preliminary reflection of the scalp and periosteum. Care is to be exercised in going through the inner table. A fine needle fitted with a syringe is then inserted through the opening for purposes of exploration. The puncture may be repeated frequently on the same patient. Puncture of the lateral ventricle may be performed from the front (Von Bergmann), the side (Keen), from behind, or, as recommended by the author, and by Kocher, Neisser, and Pollack, from above—2 cm. from the middle line and 3 cm. from the pre-central fissure. The needle is pushed downwards and backwards, the ventricle being at a depth of 5 to 6 cm. Subcutaneous drainage of the ventricles may follow this proceeding.

HENRY J. DUNBAR.

**SECTION OF THE POSTERIOR PRIMARY DIVISIONS OF THE
(646) UPPER CERVICAL NERVES IN SPASMODIC TORTI-
COLLIS. ROBERT KENNEDY, *Brit. Med. Journ.*, Oct. 3, 1908.**

SECTION and excision of a portion of the spinal accessory nerve is not successful as a means of treating severe cases of spasmodic torticollis, because in such cases the spasm is not produced merely by the muscles supplied by the spinal accessory, but also by certain muscles of the back of the neck of the opposite side acting along with the sterno-mastoid. The only certain means of abolishing the spasm is to destroy the spinal accessory which supplies the affected sterno-mastoid, and also the posterior primary divisions of the upper cervical nerves which supply the affected muscles of the back of the neck on the opposite side.

This procedure was first done by Gardner and by Keen in 1888, but the methods of exposure of the posterior primary divisions which have been described are unsatisfactory, the work being difficult to perform with precision. As the operation is not successful unless carried out completely, viz., by destruction of the upper four posterior primary divisions, it is important to have a free access to the nerves. The author has practised exposure by means of a longitudinal incision running midway between the external occipital protuberance and the external ear. Cutting in the same line the splenius capitis is divided and the outer edge of the complexus exposed. One or two of the slips of origin of the complexus are then detached and the complexus folded inwards. Free access is thus obtained to the posterior primary divisions from the first to the fifth. In an ordinary severe case the nerves are then freely excised, and the result is that the spasm is at once abolished and does not return.

The disadvantage of the operation is its destructive character; for while it removes a severe spasmodic condition, it replaces this by paralysed groups of muscles and by an area of anæsthesia. These defects are welcome substitutes for the condition from which a patient badly affected has suffered, but at the same time they are defects of the treatment which it would be desirable to avoid. In the ordinary severe case which is of long standing, any attempt to suture the nerves after division would result in a recurrence of the spasm after regeneration had occurred. In a case in which the spasm had lasted only three months, which had been under medical treatment without avail from the commencement, and in which the severity of the spasm was so extreme that surgical intervention was decided upon, the author sectioned and *immediately sutured* the posterior primary divisions of the upper five cervical nerves and dealt with the spinal accessory in the same way. At the time of publication (three and a half years) no recurrence had taken place. The advantage is that there is no paralysis of muscles or cutaneous anæsthesia. The rationale of this operation is that the abolition of the spasm permits the medical treatment of the case to be effectively carried out, so that by the time that the function returns in the muscles the cause of the spasm has been overcome. Obviously this modification of the method is only applicable to those cases of short duration and great severity in which there is hope of overcoming the cause of the spasm by further treatment, which is more effective after the spasm has been abolished by nerve section. There will, of course, always be the possibility of recurrence, even in specially selected cases, in which case, however, the operation can be repeated and the nerves destroyed.

AUTHOR'S ABSTRACT.

**A CASE OF SEVERE TRIGEMINAL NEURALGIA SUCCESS-
(647) FULLY TREATED BY EXCISION OF THE GASSERIAN
GANGLION.** E. W. HEY GROVES, *Bristol Med. Chir. Journ.*,
Sept. 1908.

A MARRIED woman of forty had had one previous attack of neuralgia on the right side of her face, lasting six months. The last attack had lasted for ten months. She had had thirteen teeth extracted and had been treated by many drugs without any benefit. Latterly the pain had been almost constant and the paroxysms very severe. Each began in the chin and spread to the cheeks, jaws, and tongue; they lasted from one to five minutes, and when under observation they recurred every five minutes to quarter of an hour. On the affected (right) side of the face the skin was

rough and excoriated and all the hair lost from the constant rubbing during the paroxysms. On February 27, 1907, the operation was performed. The right common carotid was clamped to lessen hæmorrhage and the eyelids on the right side stitched together. The usual Hartley-Krause operation was performed, the bone being removed with a trephine. The blood was removed from the depth of the wound by the use of a constant suction apparatus. After the third and second divisions had been cut and an attempt was being made to free the ganglion, such furious bleeding occurred from the cavernous sinus that the latter had to be plugged and the patient put back to bed. On March 4th the operation was completed, and proved so easy that it would suggest that the routine performance of the operation in two stages is a better method of overcoming the difficulties due to bleeding than clamping the carotid artery. A year later she was in excellent health, with no return of the pain. Over the right cheek and lips there was an area of anæsthesia to light touch (epicritic sensation), which ended at the tragus of the ear in an abrupt right angle. The corneal sensation was dulled but not lost, and there was no loss of sensation in the forehead or eyelids, showing that the ophthalmic division had escaped removal. Within the anæsthetic area she could still feel deep pressure or the prick of a pin (protopathic sensation). The mucous surface of the cheek, gums, and half the tongue had lost all sensation to touch or pain. Microscopical examination of the ganglion by Professor Michell Clarke showed no abnormality. AUTHOR'S ABSTRACT.

CONTRIBUTION TO THE STUDY OF THE ELECTRIC AND (648) OPERATIVE TREATMENT OF PERIPHERAL FACIAL PARALYSIS. (*Contributo allo studio della cura elettrica e chirurgica delle paralisi periferiche del facciale.*) FUMAROLA, *Riv. di Patol. nerv. e ment.*, July 1908, p. 289.

THE author has observed forty cases of peripheral facial palsy in the past two years.

The cases treated electrically were subjected to currents of 2-4 milliamperes for about ten minutes three times a week. In cases where there was pain in the facial nerve, both spontaneous and on pressure, the treatment was not commenced till such pain had disappeared. Cases which showed reaction of degeneration only improved relatively, and no complete cures were effected. In young persons and those in whom the paralysis was incomplete, cures were generally obtained after from two to three months of treatment. Electrical treatment was found to be useless in combating secondary contracture.

A case of facial palsy which had lasted a year, following on a revolver wound in the right ear, was submitted to surgical interference after electrical treatment had failed, and Professor Bastianelli performed a facial spinal accessory anastomosis. The electrical examination of the facial muscles before operation showed a complete loss of faradic excitability in all the affected muscles, with a notable galvanic diminution of excitability and polar change.

After about five months from the date of the operation the faradic excitability had returned. There was, however, a permanent atrophy of the upper portion of the right trapezius and the sternocleidomastoid. The muscles continued to act synergically with the facial muscles at the time of writing—a year after the operation. The only indisputable good result was a certain recovery of tonicity of the right facial muscles in repose, but there was practically no independent voluntary motion of the right face, apart from the sternomastoid and trapezius contraction. No benefit was obtained from an attempt at muscular re-education.

The author considers that we are still seriously in need of evidence as to the relative efficacy of the operations of facial hypoglossal and facial spinal accessory anastomosis.

F. GOLLA.

DIPHTHERITIC PARALYSIS AND ITS TREATMENT. (Ueber (649) diphtherische Lähmungen und ihre Behandlung.) KOHLS, *Therap. Monatsh.*, 1908, p. 329.

AFTER a sketch of the phenomena of diphtheritic paralysis, the author states that, inspired by Comby, he has employed large doses of antitoxin for this condition. Illustrative cases are given, but are too fragmentary to be of any value.

J. D. ROLLESTON.

SERUMTHERAPY IN DIPHTHERITIC PARALYSIS. (Contribution (650) à l'étude de la sérumthérapie des paralysies postdiphthériques.) SCHNEIDER et VANDEUVRE, *Progrès Médical*, 1908, p. 421.

A SOLDIER, aged 22, had an attack of severe diphtheria in May 1907, for which he received antitoxin. Forty days later palatal palsy, palpitation, and various sensory disturbances ensued. On admission to hospital on June 27 he showed generalised paralysis and amyotrophy, with hypæsthesia for all modes of sensibility.

The heart sounds were muffled and intermittent, with a tendency to embryocardia. The skin and tendon reflexes were completely absent. In the course of the following week he received five injections of antitoxin. Rapid improvement followed. By July 6 he was able to walk round his bed, and on August 6, when he was discharged, the paralysis had disappeared, but the knee and ankle jerks were still absent.

(Was the improvement in this case due to antitoxin, or, as recently suggested by the reviewer on commenting on a similar case, the effect of suggestion? (*v. Lancet*, July 25, 1908, p. 261). —J. D. R.) J. D. ROLLESTON.

SURGICAL TREATMENT OF TUMOURS OF THE SPINAL CORD.

(651) (*Beitrag zu chirurgischer Behandlung der Rückenmarkstumoren.*) FLATAU and ZYLBERLAST, *Zeitsch. f. Nervenheilk.*, 1890, Bd. 35, S. 334.

THE case was one of extramedullary tumour in the cervical region of the cord in a woman aged forty years. Fourteen weeks before admission she had sudden pain in the left foot. The pain gradually increased in intensity, and within a day it extended up the left lower limb and included the left half of the trunk (the face and upper limb being unaffected). On the third day there was pain in the right leg and in the sacral region. The pain was followed by weakness of the legs, so that after a fortnight the patient was bedridden. Four weeks before admission trembling of the legs was noticed on slight voluntary movement. Then cramp appeared, also paræsthesia in the legs. On examination the cranial nerves were normal. The vertebral spaces were tender on pressure from the lower cervical down to the upper lumbar region. The upper limbs were normal, the lower limbs were weak, especially the left leg. There was considerable rigidity of the muscles of both legs. There was knee-clonus and ankle-clonus with extensive plantar reflex on both sides. Sensory disturbances were somewhat difficult to elicit, owing to patient's deficient attention, but distinct loss was present to touch, temperature, and pain on the left side, below the third intercostal space. These changes were marked on the front of the body, but on the posterior aspect they were reversed, affecting the right lower limb and right side of the trunk from the shoulder downward. This phenomenon was suggestive of a hysterical element, but afterwards it was found to be apparently of organic origin. Sense of position was lost in the left lower limb. Incontinence of urine was occasionally present. Gradually, in the course of three or four

months' hospital treatment, the pains became more marked on the right side. The cerebrospinal fluid was under increased pressure, but free from lymphocytes. The sensory loss became practically symmetrical from the third rib downward. Ultimately paralysis of the lower limbs became complete, with total retention of urine, girdle pains around the thorax, and anæsthesia below the level of the first thoracic space posteriorly, and from the fourth intercostal space anteriorly, extending on the right side an interspace higher. The left pupil and left palpebral fissure were diminished. Operation was undertaken, and an extramedullary tumour was removed from the left side at the level of the sixth and seventh cervical vertebræ, measuring 2·3 cm. long, 1·8 broad, and 0·9 thick. Microscopically the growth was a spindle-celled sarcoma. On the day after operation there was blunting of sensibility in the left little finger and ulnar edge of the left forearm, but the sensation of the feet and of the left leg had improved. Two days after operation feeble voluntary movements reappeared in the right foot, right knee, and left foot, and the sensibility returned on the right side of the trunk. Gradual return of motor power occurred in the legs, the right recovering faster than the left. The anæsthesia disappeared from above downwards on the right side of the body, the left side clearing up much faster. After 2½ months the patient was again able to walk without support, and six months after operation the gait was practically normal. The plantar reflexes were still extensor in type.

PURVES STEWART.

DIVISION OF THE AUDITORY NERVE FOR PAINFUL TIN-
(652) **NITUS.** CHARLES A. BALLANCE, *Lancet*, Oct. 10, 1908.

THE operation was performed in January 1908 by Mr Ballance, the patient having been under Dr Ferrier's care. The illness commenced suddenly in November 1906 with deafness of the right ear, associated with vertigo, nausea, and tinnitus. The tinnitus was of a whistling or steaming character, and at its height became actually painful. The semi-circular canals were removed in November 1907, with relief to the vertigo, but without relief to the tinnitus. For many weeks after the operation the patient's condition was very grave. Dr Purves Stewart made a note on October 18, 1908, which appears in full in the *Lancet* of October 24. She still has slight intermittent buzzing tinnitus, but what she complains of now is a painful dragging sensation on the same side of the head.

AUTHOR'S ABSTRACT.

Review

"MANUAL OF PSYCHIATRY." By J. ROQUES DE FURSAC, M.D.
Translated by A. J. Rosanoff, M.D. New York: John Wiley
& Sons. London: Chapman & Hall, Limited, 1908. Price
10s. 6d. nett.

THIS manual is the authorised second American edition from a revised and enlarged second French edition. It is a compact book, obviously intended for students preparing for final examinations for degrees where psychological medicine is a subject, and for those it can be recommended as a careful compendium of the essentials of psychiatry. In such a book brevity is a necessary feature, but the authors have brought the book well up to date, and there are numerous references to the recent works of psychiatrists throughout the pages.

The book is divided into two parts. In Part I. the general principles of psychiatry are treated on conventional lines. The chapters on symptomatology are particularly well written. There is a chapter devoted to the examination of the insane, with an excellent plan of the method used by the authors in history taking, while the latter portion of the same chapter gives a short summary of psycho-therapeutics.

In the second part of the book the clinical varieties of insanity are succinctly dealt with in turn, the main etiological, symptomatic, and therapeutic features of each being briefly discussed. The classification followed is that of Kraepelin, with some slight modifications.

C. J. ROBERTSON MILNE.

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Indices

Page references to Original Articles are indicated by heavy type figures.

SUBJECT INDEX.

- ABASIA** : or Dysbasia, 113
Abdomen : Segmental Paralysis of, 247
Abducens : Isolated Paralysis of, 305 ;
 Paralysis of, after Spinal Anæsthesia,
 350
Abscess : of Brain, 720 ; Anosmia in
 Temporo-sphenoidal Abscess, 654 ;
 Bronchiectasis and, 356, 424 ; Otitic,
 40 ; Fatal, 180 ; Due to Typhoid
 Bacillus, 249 ; Frontal, 492
Aconitine : Action on Nerve-Fibres, 94
Acromegaly : and Syringomyelia, 108 ;
 with Osteo-Arthropathies and Para-
 plegia, 256 ; Osseous Plaques of Pia
 Arachnoid and Pain in, 661 ; Tabes
 Associated with Trophic Changes sug-
 gesting, 607
Adiposis Dolorosa : 431 ; in Dementia
 Præcox, 315
Adrenalin : Action on Central Nervous
 System of Rabbit, 474
Agraphia : *vide* Aphasia
Alcohol : Central Nervous System in
 Alcoholised Rabbits, 28 ; Delirium
 Tremens after Withdrawal of, 47,
 200 ; in Etiology of Insanity, 48, 511 ;
 Deep Injections of, for Trifacial Neur-
 algia, 131 ; Injections for Trigeminal
 Neuralgia, 202 ; Influence of, on Fatigue,
 446 ; Korsakow's Disease, 371, 441,
 627 ; and Mental Disease, 626
Alcoholism : Neurofibrils in Chronic, 97 ;
 Alcoholic Neuritis, 98 ; Insanities, 123 ;
 Delirium Tremens, 200 ; Alcoholic
 Epilepsy, 258 ; Post-Delirious Stupor
 in, 259 ; Complications of, 260 ; and
 Insanity, 268 ; Asylum Treatment in,
 319 ; Cerebral Cortex in, 346 ; Chronic,
 in a Child, 366 ; Chronic Non-moral
 Alcoholics, 512
Alexia : *vide* Aphasia
Allochiria, 294
Amaurotic Family Idiocy, 186
Amblyopia : Toxic, 307 ; after Accessory
 Sinus Suppuration, 437
Amyotonia Congenita, 481
Amyotrophic Lateral Sclerosis in Boy
 of 16, 99
Anæsthesia : Spinal, 204, 350 ; Total,
 500
Anchylostomiasis : Mental Disorders of,
 268
Aneurism : of Larger Cerebral Arteries,
 36 ; of Anterior Cerebral Artery, 449 ;
 of Internal Carotid Artery and Cavern-
 ous Sinus, 462 ; of Sylvian Artery,
 496 ; of Cerebral Vessels, 720
Ankle Clonus : *vide* Reflexes
Anorexia Nervosa in Children, 115
Anosmia in Temporo-Sphenoidal Ab-
 scess, 654
Aphasia : 654 ; with Integrity of Left
 Third Frontal Convolution, 36 ; Aphe-
 mia, 655 ; Alleged Word Deafness in
 Motor, 505 ; Symptoms and Basis of
 Word Deafness, 619 ; Pure Word Deaf-
 ness in Hysteria, 113 ; Congenital
 Word Blindness, 510 ; Word Blindness
 with Agraphia, 509 ; Paraphasia, 621 ;
 Conduction Aphasias, 507 ; and Uræmia
 Hemiplegia, 560 ; in Functional
 Psychoses, 428 : *vide* Apraxia and
 Asymboly
Aphemia : Case of Pure, 655
Aphonia : Organic and Functional, 113
Apoplexy : Tremor following, 180
Appendicitis, Hysterical, 661
Apraxia : 658 ; Motor, 357 ; Bilateral
 Motor, with Apraxia of Eye Muscles,
 etc., 657 ; and Dementia Præcox, 666 ;
 in Juvenile General Paralysis, 666 ;
 and Aphasia, 508 ; and Left Hemi-
 plegia, 511, 560 ; Eupraxia, 622
Arsenic : In Treatment of Chorea Minor,
 204
Arteries : Occlusion of Posterior Inferior
 Cerebellar, 557 : *vide* Aneurism
Arterio-sclerosis : 119 ; Symptoms due
 to Coronary, 46 ; Atrophy of Cere-
 brum, 54 ; Nervous Manifestations of,
 262
Arthropathy : in Acromegaly, 256 ;

- Lesions in Anterior Horn Cells in Nervous, 594**
Arthrodesis and Tendon Transplantation in Paralytic Conditions, 317
Articulation of Consonantal Sounds in School Children, 243
Ascending Paralysis: 246, 710; Acute, of Syphilitic Origin, 275
Astereognosis, 502; in Disease of Post-Central Gyrus, 379
Asthenia: Constitutional, 113
Asymby: Cortical Localisation of, 717; and Aphasia, 107
Ataxia: Acute, 111; Marie's Hereditary Cerebellar, 420
Atropin: Action of, on Autonomic Nervous System, 590
Auditory Tracts, 402
- BABINSKI, Sign of: vide Reflexes**
Barium Chloride: Action of, on Fowl's Muscle, 705
Basedow's Disease: vide Exophthalmic Goitre
Bell's Phenomenon, 619
Beri-Beri: Nissl's Stain in, 406
Blepharospasm: Psychological Nature of, 722
Blood: Changes in Mental Diseases (Review), 205; in Exophthalmic Goitre and Thyroidism, 721
Brachial Plexus: Complete Radicular Paralysis of, with Oculo-pupillary Phenomena, 624
Brain: Anatomy: Perivascular Corpuscles in Substance of, 20; New Origin of Peduncular Bundle of Türk, 91; Induseum Griseum Corporis Callosi, 159; Nucleus Ruber Tegmenti, 159; Origin of Superior Facial, 162; Floor of Fourth Ventricle, 235; Structure of Grey Matter, 283; Arcuate Nuclei and External Anterior Arciform Fibres of Medulla Oblongata, 471; Mechanism and Function (Review), 519; Cerebello-Olivary Fibres, 544; Origin of Facial Nerve, 685; Three Chinese Brains, 697; Brain Matter, 697; Projection Fibres of Occipital Lobe, 698
Physiology: Relation between Labyrinth and Eye, 92; Afferent Impulses and Fatigue of Vasomotor Centre, 94; Functions of Corpora Striata, 254; Reflex Excitability after Cerebral Anæmia, 344; Supranuclear Auditory Tracts, 402; Pons and Corpora Quadrigemina, 585; Centre for Submaxillary Gland, 588
Pathology: Postcentral Cortex in Tabes, 5; Traumatic Lesion of Pons and Tegmentum, etc., 26; Lenticular Nucleus, 26; Medullary Sheaths in Cortex of General Paralytics, 96; Heterotopia of Nucleus Arcuatus, 168; Cell Findings in Soft Brains, 168; Cortical Changes in Tumours, 169; Experimental Lesions at Base, 169; Cortex in Alcoholics, 346; Senile Cerebellum, 346; Contributions to, 404; Forced Movements in Central Lesions, 549; Sequel to Aseptic Lesions of, 642; Areas of Disease in Cortex in Multiple Sclerosis, 706; Cortical Localisation of Asymby, 717
Clinical: Aneurisms of Larger Cerebral Arteries, 36; Otitic Brain Abscess, 40; Traumatic Softening of Corpus Callosum, 51; Treatment of Arteriosclerotic Atrophy of Cerebrum, 54; Symptoms of Atrophy of Occipital Lobe, 106; Fatal Abscess of, 180; Gliotic Cyst of Right Superior Parietal Lobule, 182; Intracranial Abscess from Typhoid Bacillus, 249; Hæmorrhage into Pons Varolii in Eclampsia, 250; Dissociation of Colour-Sense in Focal Disease, 291; Pontile Hæmorrhage, 295; Eye-Movements in Cerebellar Irritation, 304; Cerebellar Abscess, 356; Bronchiectasis and Cerebral Abscess, 356, 424; Focal Symptoms in Diffuse Disorders, 359; Cerebellar Atrophy, 359; Disease of Post-central Gyrus, with Astereognosis, 379; Traumatic Abscess of Frontal Lobe, 492; Integrity of Sensation with Lesion of Left Parietal Lobe, 501; Cerebral Complications of Nasal Origin, 645; Anosmia in Temporo-sphenoidal Abscess, 654; Bulbo-pontine Softening, 612; vide Abscess, Aneurism, Tumour, etc.
Bromide: Large Doses of, in Nocturnal Petit Mal, 202
Bronchiectasis and Cerebral Abscess, 356
Bulbar Paralysis: Acute, 421; in Syringomyelia, 172, 555, 652
- CAFFEINE: Action of, 640**
Caisson Disease: Lesions of Spinal Cord in Experimental, 479
Calmette Reaction, 117
Cardiac Mechanism after Isolation from Extrinsic Nerve Impulses, 93
Catatonia: Syringomyelic Lesion in Stupor, 26; in Dementia Præcox, 198
Cauda Equina: Tumour of, 287; Unilateral Cauda Equina Syndrome, 611; vide Cornus Medullaris
Cavernous Sinus: Aneurism of, 462; Thrombosis of, 720

- Cerebellum**: Senile, 346; Classification of Diseases of, 420; Occlusion of Posterior Interior Cerebellar Artery, 557; Abcess of, 356; Eye-Movements in Irritation of, 304; Atrophy of, 359: *vide* Tumours
- Cerebellar Ataxia**, 420
- Cerebro-spinal Fluid**: in Tubercular Meningitis, 39; in Paresis, 121; Cytological Study of, and Diagnostic Value in Psychiatry, 207; Cytological Study of, 297; Lymphocytosis of, in Lues Hereditaria Tarda, 297; Cholin in, 298; Typhoid Bacilli in, 491, 597; in General Paralysis, 635; in Anterior Poliomyelitis, 651; in Diphtheritic Pseudo-Tabes, 712; Cytological Examination of, 713
- Cervical Ribs**: and Atrophy of Intrinsic Hand Muscles, 191; Symptoms due to, 312
- Chateaubriand**: Medical Study of, 573
- Cheiromegaly and Syringomyelia**, 172
- Cholesterin**: Power to Neutralise Hæmolytic Action of Lecithin and Specific Serums, 480
- Cholesterol Fluid Crystals**, 638
- Cholin in Cerebro-spinal Fluid**, 298
- Chorea**: With Double Optic Neuritis, etc., 44; Treatment of Chorea Minor, 132; Psychical Disturbances in Sydenham's, 188; Certain Pupillary Signs in, 191; Treatment by Arsenic, 204; Chronic Progressive, 498; Motor Phenomena of, 499; Mental State in, 563
- Circumflex Nerve in Diphtheria**, 170
- Cocaine**: Affinity of Spinal Cord for, 94
- Colour-sense**: Dissociation of, in Focal Brain Disease, 291; Splitting off of, 561
- Congress of Alienists and Neurologists of France**, 624
- Consciousness**: Post-traumatic Transitory Disturbances of, 427
- Conus Medullaris**: Diseases of, 653; Epiconus Symptom-complex in Cerebro-spinal Syphilis, 77
- Corpora Striata**: Functions of, 254
- Corpus Callosum**: Traumatic Softening of, 51
- Crime**: and Insanity, 125, 126, 127, 313, 316; New Classification of Criminals (Review), 444
- Cyanide of Potassium**: Disease of Primary Motor Neurones from Poisoning by, 555
- Cytodiagnosis**: in Practical Medicine, 264
- DE QUINCEY**: Psychological Study of (Review), 273
- Defectives**: Co-operation of Alienist in Case of, 373
- Degenerates**: Paranoid Symptom-complexes in, 196
- Delirium Tremens**: 200; after withdrawal of Alcohol, 47
- Dementia Paralytica**: *vide* General Paralysis
- Dementia Præcox**: Catatonic Form of, 198; Adiposis Dolorosa in, 315; Complexes and Etiology in, 367; Idiocy and, 368; Cases, 569; in India, 569; Final Stages of, 570; Eye Syndrome of, 570; Bulbo-Cavernous Reflex in, 665; Case of Paranoid Form, with Autopsy, 665; Apraxia and, 666; Neurofibrils in Senile, 97
- Dengue**: Spondylitis Infectiosa after, 115
- Dercum's Disease**: *vide* Adiposis Dolorosa
- Diphtheria**: Experimental, 29; Pathogenesis of Paralysis and Heart Failure in, 116; Paralysis in, 170; Neuritis of Left Circumflex Nerve in, 170; High Incidence of Nervous Complications in, 664; Treatment of Diphtheritic Paralysis, 728 (2); Cerebro-spinal Fluid in Diphtheritic Pseudotabes, 712
- Disseminated Sclerosis**: 31; Sacral Type of, 32; Acute or Disseminated Myelitis, 33; or Cerebro-spinal Syphilis, 34; Acute Retrobulbar Neuritis and, 300; commencing with Failure of Vision, 424; Course and Progress in, 521; Pathological Anatomy of, 706
- Double Personality after Hæmorrhage**, 296
- Dysbasia**, 113
- Dyspraxia**: *vide* Apraxia
- EARS**: Associated Movement of Eyes and, 331
- Eclampsia**: Treatment of, 53; Hæmorrhage into Pons Varolii as Cause of Death in, 250
- Electrical Treatment**: of Facial Tic-Douloureux, 52
- Encephalomyelitis**: Acute, 354; Poliomyelitis in Boy of Three, 492
- Epiconus Symptom-complex in Cerebro-spinal Syphilis**, 77
- Epilepsy**: Etiology of, 721; Heredity in, 29; Epileptoid Convulsions in Typhoid, 37; Borderland of (Review), 134; Nocturnal Petit Mal curable by Large Doses of Bromide, 202; Alcoholic, 258; Operation as Therapeutic Measure in, 271; Paralysis Agitans in, 315; Corpuscular Resistance of Serum in, 365; Jacksonian, 425; Mechanism

- of Gliosis in, 497 ; so-called Idiopathic Form, 498 ; and Chronic Delusional Insanity, 513 ; Colony and Bromide Treatment of, 563
- Eupraxia**, 622
- Exophthalmic Goitre**: 363 ; with *Myasthenia Gravis*, 229 ; Treatment of (Review) 327 ; treated by Thyroidectomy, 362 ; Heart Failure in, 566 ; X-Ray Treatment of, 566 ; Antithyroid Treatment of, 659 ; Blood in, 660, 721 ; Surgical Treatment of, 669
- Exophthalmos**: Intermittent, 435
- Eyes**: Action of X-Rays during Development of, 242 ; Conjugate Deviation of Head and, 306 ; Associated Movement of Ears and, 331 ; Headache and Eye-strain, 428 ; in General Paralysis, 441 ; in *Dementia Præcox*, 570 ; Apraxia of Eye Muscles, 657 ; Movements of, in Irritation of Cerebellum, 304
- Eyelids**: Synchronous Movements of, with Tongue and Lower Jaw, in certain Diseases, 35 ; Partial Ptosis with Exaggerated Involuntary Movement of Affected, 337
- FACIAL Nerve**: Electrical Treatment of Tic-Douloureux, 52 ; Pathological Anatomy of Peripheral Paralysis and Hemispasm, 167 ; Peripheral Palsy, 171 ; Theory of Paralysis of, 362 ; Difference between Central and Peripheral Paralysis of, 484, 545 ; Origin of, 162, 685 ; Otalgia as Sensory Affection of Seventh Cranial Nerve, 187 ; Electric and Operative Treatment of Peripheral Facial Paralysis, 727 ; Bell's Phenomenon, 619
- Family Diseases**: Relation to Premature Physiological Senescence, 595
- Family Spastic Paraplegia**, 352
- Fatigue**: Measurements in 64 School Children, 20 ; in Frog's Nerve, 166 ; Measurement of Intellectual, by the *Æsthesiometer*, 407 ; Influence of Alcohol, etc., on (Review), 445 ; of Nerves, 472 ; Action of Active Suprarenal Principle on Muscular, 547
- Freud's Psycho-Analytic Treatment of Insanity**, 192, 193, 572
- GALVANIC Phenomenon** in Normal and Insane, 122
- Ganglia**: Plasticity and Amœboidism of Cells of Sensory, 28 ; Hypospinal Micro-Sympathetic, 234 ; Structure of Spinal, 468 ; Poliomyelitis Posterior of Genuate, 485 ; Herpetic Inflam-
- mations of Genuate, etc., 648 ; Excision of Gasserian in Trigeminal Neuralgia, 726
- Gangrene**: Multiple Relapsing, in Arms and Foot, 567
- Gasserian Ganglion**: Excision of, in Trigeminal Neuralgia, 726
- Gastric Juice** in Psychopathological Conditions, 51
- General Paralysis**: with Cerebral Syphilis, 46 ; Patchy Atrophy of Medullary Sheaths in Cortex in, 96 ; Nerve Fibrils in, 97 ; Progressive (Review), 134 ; Changes in Spinal Cord in, 168 ; Statistical Study of, 196 ; Clinical Course of, 196 ; in Children, 266, 625, 666 ; Three Years after Syphilitic Infection (2), 267 ; in Senile Period, 268 ; Syphilitic, 293 ; Peripheral Nerves in, 345 ; Eye Findings in, 441 ; Serum Reaction of Syphilis in, 572 ; Frontal Tumour Simulating, 560 ; Cerebro-spinal Fluid in, 121, 635
- Genuate Ganglion**: Poliomyelitis Posterior of, 485 ; Herpetic Inflammations of, 648
- Glands**: Polyglandular Syndromes and Opothrapy, 669
- Gonorrhœa**: Meningitis following, 174 ; Acute Gonorrhœal Inflammation of Labyrinth, 647 ; Gonorrhœal Neuritis of Auditory, 649 ; Optic and Oculomotor Neuritis following, 485 ; Spondylitis in, 193
- Graves' Disease**: *vide* Exophthalmic Goitre
- HÆMORRHAGE**: *vide* Brain and Spinal Cord
- Headache**: from Pathological Conditions of Nose, etc., 366 ; and Eye-Strain, 428
- Hemicrania**: Urinary Constituents in, 35 : *vide* Megrim
- Hemiplegia**: in Typhoid, 37, 177 ; Muscular Strength in, 253 ; Pseudo-Hysterical, 253 ; Side affected by Hysterical, 293 ; Hysterical, 357 ; Palato-Laryngeal, 493 ; with Unilateral Optic Atrophy, 494 ; Word-Blindness with Agraphia in Left-Handed Hemiplegic, 509 ; Dyspraxia with Left-Sided, 511, 560 ; Relative Eupraxia in Right, 622 ; following Scarlet Fever, 530 ; Uræmic, and Aphasia, 560 ; Precocious, in Secondary Syphilis, 659
- Heredity**: 120 ; in Diseases of Nervous System, 29
- Herpes**: Gluteal, after Lumbar Puncture,

- 38; in Epidemic Cerebro-spinal Meningitis, 38, 103; Neuritis secondary to, 350; and Mumps, 560; Herpetic Inflammations of Geniculate Ganglia, 485, 648; of Membrane Tympani, due to Zosteroid Affection of Petrosal Ganglion, 619
- Hiccough: in Syringomyelia, 652
- Homosexuality: Grouping of, 372; Diagnosis of, 373
- Hydrocephalus, Meningococcal, 418
- Hypnosis: State of Brain during, 592
- Hypotonia, 409
- Hysteria: Definition of, 41; and Litigious Insanity, 48; Pure Word Deafness, 113; Severe Briquet Attack, 114; with Periodic Melancholia, 124; Pseudo-Tetany and Peculiar Vaso-Motor Disturbances, 192; Freud's Psycho-Analytic Treatment of, 193; Pseudo-Hysterical Hemiplegia, 253; Ankle Clonus in, 263; Psycholeptic Attacks in, 264; Diagnosis of Organic from Functional Disease, 284; Hemiplegia in, 293, 357; in Children, 297; Immobility of Pupils, 303; Major Symptoms of (Review), 374, Trophic Disorders in, 426; Dissociation of Reflexes in, 426; Freud's Theory of, 427; Hysterical Mutism, 504; Pseudo-Appendicitis Hysterica, 661; Aphonia, 113; Sign for Detecting Functional Paresis in Lower Extremities, 662; Non-traumatic Pseudopastic Paresis, 112; Revision of, 616; Torticollis Mentalis (Hystericus), 618
- IDIOTCY: Neurofibrils in Microcephalic, 97; Amaurotic Family, 186; and Dementia Præcox, 368; Secondary to Diseases of Cerebral Vessels, 371
- Indoxyluria: in Mental Diseases, 50
- Infantile Paralysis, 245; Scoliosis in, 286; Resembling Meningitis, 413; Use of Silk Ligaments in, 515: *vide* Poliomyelitis
- Influenza: Psychoses of, 439
- Insanity: Alcohol in Etiology of, 48, 511; Hysteria and Litigious, 48; Galvanic Phenomena and Respiration in, 122; Alcoholic, 123; Hysteria with Periodic, 124; Criminal Responsibility in, 125; and the Penal Law, 127; Alcoholism and, 268; Pains in Manic-Depressive, 268; Opsonic Index to Various Organisms in, 270; Paralysis Agitans in, 315; Forced Speech in Manic-Depressive, 369; Term "Manic-Depressive Insanity," 369; Simulation of, 373; Adolescent, 440; Chronic Delusional, and Epilepsy, 513; Modern Care and Treatment of, 514; Pupillary Phenomena in, 568; of Maupassant, 574; Thyroid Gland in, 664; Simulation of (Review), 671, 313
- JAW: "Jaw-winking Phenomenon," 337; Synchronous Movements of Tongue and Lower Jaw, 35
- Jews: Mental Diseases among, 443
- KERATITIS: Bacteriology of Neuro-pathic, 185
- Kernig's Sign, 40
- Korsakow's Disease, 371, 441, 627
- LABYRINTH: Destruction of, 646; Acute Gonorrhœal Inflammation of, 647; Relation between Eye and, 92
- Landry's Paralysis, 246 (2), 375, 710
- Larynx: Parathyreogenic Laryngospasm, 433; Laryngeal Crisis in Tabes, 652; Palato-laryngeal Hemiplegia, 493; Sensory Fibres in Recurrent Laryngeal Nerve, 238; Recurrent Paralysis, 365
- Lenticular Nucleus, 26
- Leptothrix Infections, 631
- Locomotor Ataxia: *vide* Tabes Dorsalis
- Lumbar Puncture: Gluteal Herpes following; 38; in Cerebro-spinal Meningitis, 53; in Tuberculous Meningitis, 175; Technique of, in Children, 177; in Optic Neuritis, 205; in Meningeal Forms of Typhoid, 557; Paralysis after Rachistovainisation, 351
- Lunatic Asylums: Construction of, 316
- MALINGERING: Sign for Detection of, 662
- Maupassant: Insanity of, 574
- Melancholia: and Manic-depressive Insanity (Review), 61; Recognition and Treatment of (Review), 64; Periodic, with Hysteria, etc., 124; Homicidal, 440
- Meningitis: Serous Spinal, 650, 710; Acute Aseptic, 714; Acute, of Convexity, 416; Pyæmia and, 631; Cerebral and Cerebro-spinal during Puerperium, 599; in Infantile Paralysis Simulating, 413; Systematic Lumbar Puncture in, 53; Surgery of Otogenic, 668; in Mumps, 249 (2); in Typhoid, 597 (2); Gonorrhœal, 174, 598; Acute Syphilitic, 174, 290, 419, 599; Tuberculous, 39, 175, 291, 598, 715; Epidemic Cerebro-spinal, 39, 53, 103, 104, 288, 289.

- 353, 417, 418, 489; Serum Treatment of, 352, 417, 600, 715, 716: *vide* Cerebro-spinal Fluid
- Meningococcus Infections**, 100; Hydrocephalus from, 418
- Mental Diseases**: Indoxyluria in, 50; Obstruction in Nose and Throat as Cause of, 117; and Crime, 125, 126, 127; Prognosis in Cases showing Feeling of Unreality, 199; Changes of Blood in (Review), 205; Alcohol in Etiology of, 511, 626; Mental Defective in Prison, 575; Calvin's Disease, 437; Medical Study of Chateaubriand, 573; De Quincey, 273; in Jews, 443
- Mental Disturbances**: in Vasomotor Neuroses, 296; Cranial Trauma and, 369; in Anchylostomiasis, 268; in Unilateral Nasal Obstruction, 627
- Menthol Poisoning**, 567
- Meralgia**: Anterior Paræsthetic, 245
- Mercury Treatment of Metasyphilis of Nervous System**, 127
- Methods**: New Selective Stain for Nervous System, 91; Simplification of Nissl's Stain, 406; Principles underlying Weigert's, 638; for Staining Myeline in Nerve Fibres of Brain and Cord, 682; for Demonstration of Internal Reticular Apparatus of Nerve Cells, 700
- Microcephaly**: Neurofibrils in, 97
- Middle Ear Disease**: Chronic Suppuration, complicated by Tumour of Pons, 558
- Migraine**: with Melancholia, 124; and Treatment of the Eyes, 565; an Occupation Neurosis, 618; Urinary Constituents in, 35
- Monoplegia**: Crural, 358
- Movements**: of Eyelids, Tongue, and Lower Jaw in Certain Diseases, 35
- Multiple Sclerosis**: *vide* Disseminated Sclerosis
- Mumps**: Meningitis in (2), 249; Herpes Zoster and, 560
- Muscles**: Inhibition, from Excitation of Ninth Spinal Nerve of Frog, 24; Strychnine and Reflex Inhibition of Skeletal, 24; End-Plates of, after Section of Nerves, 95; Growth in Efficiency after Age of Fifty, 132; Contraction of, 166; Internal, in Oculo-Motor Paralysis, 184; Atrophy of Intrinsic Hand, and Cervical Ribs, 191; Reciprocal Innervation of Antagonistic, 403; Contraction in Strychnine Poisoning, 473; Action of Barium Chloride on, 705
- Muscular Dystrophy**: Spinal Changes in, 137; Are there "Formes Frustes" of?, 483; Case of Old Myopathy, 349
- Muscular Movements**: Co-ordination of, in Central Nervous System, 702
- Myasthenia Gravis**, 708; Pathology of, 408; with Exophthalmic Goitre, 229
- Myasthenia**: Pseudo-, of Toxic Origin, 1; Experimental Myasthenic Reaction in Frog, 150
- Myelitis**: Disseminated, or Acute Multiple Sclerosis, 33
- Myeline Forms**, 638
- Myelomalacia**: Traumatic Cervical, 711
- Myopathy**: Case of Old, 349: *vide* Muscular Dystrophy
- Myositis**: Progressive Ossifying, in Boy æt. Eleven, 348
- Myxœdema**: Incomplete, 565
- NERVES**: Muscular Inhibition from Excitation of Ninth Spinal, 24; Physical, Chemical and Electrical Properties of, 25; End-Plates of Muscle after Section of, 95; Freezing of Frog's, and their Fatiguability, 166; Regeneration in Peripheral Segment of, 283; Otalgia, as Sensory Affection of Seventh Cranial, 187; Cochlear, in Internal Auditory Meatus, 236; Sensory Fibres in Recurrent Laryngeal, 238, 365; Neuritis of Circumflex in Diphtheria, 170; Neuritis of Auditory, 255; Stretching of Median in Spasmodic Contraction of Finger, 271; Regeneration in Peripheral Segment of, 283; Galvanic Reactions of Auditory, 308; Peripheral, in General Paralysis, etc., 345; Axon Bifurcation in Regenerated, 402; Injuries of Vagus, 434; Fatiguability of, 472; Conductivity of, at Increased Temperatures, 472; Degenerations following Injuries to Posterior Roots of 7th Cervical, 643; Clinical Appearances in Lesions of Vagus Recurrent and Oculomotor, 645; Gonorrhœal Neuritis of Auditory, 649; Rate of Transmission in Human Medullated, 704; Neuritis of Deep Palmar Branch of Ulnar, 709; Section of Posterior Primary Divisions of Upper Cervical, in Spasmodic Torticollis, 725; Division of Auditory, for Painful Tinnitus, 730; Inhibitory Fibres in Peripheral, 588; Supposed Existence of Vaso-constrictor Fibres in Chorda Tympani Nerve, 589; Origin of Facial, 685; Cutaneous Branches of Posterior Primary Divisions of Spinal, 699
- Nerve Cells**: Reticular Apparatus of Golgi-Holmgren, 90; Fibrils and Fib-

- rillogeous Substance in Ganglion, 160; Studies in, 233; Neurofibrils of Motor Ganglion, 343; of Electric Lobe of Torpedo Ocellata, 471; Method for Demonstration of Internal Reticular Apparatus of, 700; of Locus Cœrulus and Substantia Nigra, 701
 Nerve Centres: Cerebro-spinal (Review), 56; Functions of (Review), 327; First Phases of Development of, in Vertebrates, 698
 Nerve Endings: in Electric Organ of Torpedo Ocellata, 471
 Nerve Fibres: Action of Aconitine on, 94; Living Developing, 241; Course of Cerebello-Olivary, 544; Inhibitory, in Peripheral Nerves, 588; Vaso-constrictor, in Chorda Tympani Nerve, 589; Vaso-Dilator, in Depressor Reflexes, 589; Primary Lesions of, in Uremia, 707
 Nerve Fibrils: in Dementia Paralytica, Chronic Alcoholism, etc., 97; in Ganglion Cells of Vertebrates, 160; Conducting Function of, 242; in Processes and Cell-Body of Motor Ganglion Cells, 343
 Nervous Diseases: Obstruction in Nose or Throat as Cause of, 117; Trauma in Etiology, 117; Favourable Influence of Occupation in, 201; Diagnosis of Organic from Functional, 284
 Nervous System: Central, in Alcoholised Rabbits, 28; Central, in Experimental Diphtheria, 29; Report on Anatomy of (Review), 57; Tumours of (Review) 321; Action of Adrenalin on, 474; Diseases of (Review), 519; Structure and Function of Autonomic, 544; Diseases of the (Review), 675; Changes in, after Stovaine Anæsthesia, 705; Action of Nitrites on Autonomic Nervous System, 590
 Nervousness and Nutrition in Childhood, 708
 Neuralgia: and Treatment, 52, 54; Treatment of Trigeminal by Injection of Osmic Acid, 55; Deep Injections of Alcohol for Trifacial, 131; Alcohol Injections for Trigeminal, 202; Excision of Gasserian Ganglion, in Trigeminal, 726
 Neurasthenia: in the Young, 272; Diagnosis and Treatment of, 310; Blood Pressure in, 564; Auto-Suggestion in, 564
 Neuritis: Alcoholic, 98; Diphtheritic, of Left Circumflex Nerve, 170; Lumbar Puncture in Optic, 205; Peripheral, resembling Tabes, 244; of Auditory Nerve, 255; Acute Retro-bulbar, and Multiple Sclerosis, 300; Secondary to Zona, 350; Optic and Oculomotor, following Gonorrhœa, 485; of Ulnar Nerve from Deformity of Elbow Joint, 486; Gonorrhœal, of Auditory Nerve, 649; Amyotrophic Polyneuritis, 487; in Scarlatinal Rheumatism, 649; Double Optic, following Varicella, 618; Optic, in Chorea, 44; Occupation, of Deep Palmar Branch of Ulnar Nerve, 709
 Neurofibrils: *vide* Nerve Fibrils
 Neuroglia: Functions of, 587
 Neurone Theory: Present Position of, 234
 Neurones: Vaso-motor in Shock, 163
 Neuroses: Freud's Sexual Theory of, 192; Mental Disturbances in Vaso-motor, 296; Migraine, an Occupation Neurosis, 618; Attention Neurosis, 723
 Nitrites: Action of on Autonomic Nervous System, 590
 Nose: Disturbance of Psychic Functions in Unilateral Obstruction of, 627; Cerebral Complications of Nasal Origin, 645; Obstruction in, causing Nervous and Mental Disease, 117
 Nutrition and Nervousness in Childhood, 708
 Nystagmus: Influence of Rotatory Movements on, 306; Mechanism of, 359; Reflex, in Diagnosis of Condition of Vestibular Apparatus, 436
 OCCUPATION: Favourable Influence in Nervous Disorders, 201; Neuritis of Ulnar Nerve, 709
 Ocular Movements: Paralysis of Upward Associated, 361; Partial Ptosis with Exaggerated Involuntary Movement of Affected Eyelids, 337; Conjugate, of Eyes and Head, 306
 Oculo-motor Paralysis: without Involvement of Internal Muscles, 184, 305; Clinical Appearances in, 645; following Gonorrhœa, 485
 Ocular Nerves: Paralysis of Abducens, 305, 350: *vide* Eyes, ocular movements and oculo-motor paralysis
 Œdema: of Optic Disc in Angioneurotic, 257; Persistent Hereditary, of Lower Extremities, 663
 Ophthalmia: Sympathetic, 436
 Ophthalmic Reaction: to Tuberculin, 117
 Opsonic Index: to Various Organisms in Control and Insane Cases, 270
 Optic Atrophy: Unilateral, with Hemiplegia, 494; and Tower-shaped Skull, 550
 Optic Neuritis: in Chorea, 44; Lumbar

- Puncture in, 205; after Gonorrhœa, 485; after Varicella, 618
 Orientation: Disorders of, 112
 Otagia: Sensory Affection of Seventh Cranial Nerve, 187
 Otitis: Sinus Thrombosis after Purulent, 181
- PAIN:** Subcutaneous Injections of Air for Relief of, 52
 Palato-Laryngeal Hemiplegia, 493
 Paralysis: Arthrodesis and Tendon Transplantation in, 203, 317; after Rachistovainisation, 351
 Paralysis Agitans: Parathyroid Gland for, 131; in Insane Epileptic, 315; Symptomatology of, 615
 Paralytic Dementia: *vide* General Paralysis
 Paranoia: in Degenerates, 196
 Paraplegia: due to Intra-medullary Lesion, 173; with Acromegaly, 256; Family Spastic, 352; Acute, after Anti-Rabic Inoculation, 415
 Parathyroid Gland: for Paralysis Agitans, 131; and Laryngeal Spasm, 433
 Paresis: Non-traumatic Pseudo-spastic, 112: *vide* General Paralysis
 Parkinson's Disease: *vide* Paralysis Agitans
 Periarthritis Nodosa, 193
 Petrosal Ganglion: Herpes, due to Zosteroid Affection of, 619
 Petrol: Fumes causing Pseudo-Myasthenia, 1
 Pituitary: Development of Mammalian, 340; Histological Appearances of Mammalian, 341; Posterior Lobe of, 343; Physiological Action of Extracts of, 344; Hypertrophy of, after Excision of Thyroid, 546; Physiology of, 641: *vide* Tumours
 Plexus Palsy, 624
 Poliomyelitis: Acute Anterior, 30; Anterior, in Adult, 98, 605; Acute, and allied Diseases, 99; Recent Epidemic, 171; Acute, with Diplococcal Infection of Spinal Sac, 245; Scoliosis in, 286; Orthopædic Therapy of Acute Anterior, 318; Acute (Review), 324; Epidemic Acute (Review), 326; Heine-Medin Disease (Review), 324; Posterior, of Genuiculate Ganglion, 485; Silk Ligaments in, 515; Anterior, of Specific Origin, 554; 76 Cases of Acute Anterior, 554; Clinical Picture of, caused by Disease of Primary Motor Neurones, 555; Cerebro-spinal Fluid in Anterior, 651; Resembling Meningitis, 413; in Massachusetts in 1907, 604; Epidemic, 604; Nerve Anastomosis in, 630; Epidemiology of Acute, 602; Acute, following Tonsillitis, 709; Chronic Anterior, 709
 Polioencephalomyelitis, 354, 492
 Polyarteritis Acuta Nodosa, 193
 Polyneuritis: Amyotrophic, 487
 Pons: Hæmorrhage into a cause of Death in Eclampsia, 250; Hæmangioma in, 352
 Protagon: Chemical Composition, etc., 164
 Pruritus: in Tabes, 100, 607
 Pseudo-bulbar Paralysis, 179, 357; with Loss of Voluntary Respiration, 178
 Psychasthenia, 269, 310
 Psychiatry: Psychopathic Intoxication, 47; Sexual Traumata as Form of Infantile Sexual Activity, 50; Secretion of Gastric Juice in Psychopathological Conditions, 51: Freud's Theory and its Significance in Kraepelin's Manic-depressive Insanity (Review), 59; Obsessional States, 123; Cerebro-spinal Fluid in, 307; Outlines of (Review), 272; Insanity, Simulation and Criminality, 313; Expert Evidence in Criminal Proceedings, 316; Co-operation of Alienist in Case of Defectives, 373; "Zwang" Phenomena, 438; Mental Diseases among Jews, 443; Protection of Society from Criminal Lunatics, 629; Abolition of Use of Tobacco in Lucca Asylum, 629; Manual of (Review), 731
 Psychology: of Neurotic Symptoms (Review), 58; Study of Thomas de Quincey (Review), 273; Elements of (Review), 319; A Mind that found Itself (Review), 328; Alfred de Musset (Review), 329; Crowd Suggestion and Psychical Epidemics (Review), 330; The Moltke Case (Review), 330; Bismarck im Lichts der Naturwissenschaft (Review), 376; Co-conscious Ideation, 406; Coloured Thinking, 475; Psycho-Galvanic Reactions from Co-conscious Ideas in Multiple Personality, 478; Articulatory Capacity for Consonantal Sounds in Children, 243, 548; Ideas of Children, 549; Classification of Methods, 549; Calvin, 437; Rousseau, 437; Chateaubriand, 573
 Psychopolyneuritis: Chronic, 371
 Psychoses: Clinacteric, 123; Classification of, 195; Acute Traumatic, 201; Peripheral Nerves in, 345; Speech Disturbances in Functional, 428; of Influenza, 439; Combined, 513;

Psycho-Analytic Method and "Abwehrneuropsychosen" of Freud, 572 ;
Hypnoidization in Treatment of Functional, 629
Psychotherapy : Various Forms of, 55
Ptosis : *vide* Ocular Movements
Puncture of Brain, 724
Pupils : in Chorea, 191 ; Sensitiveness to Light and Size of, 239 ; Diagnostic Meaning of Symptoms in, 265 ; Physiology and Pathology of Movements of, 301 ; Eserin in Disorders of, 303 ; Peculiar Phenomenon and Hysterical Immobility of, 303 ; Eye-Movements in Cerebellar Irritation, 304 ; Diagnostic Value of Immobility and Sluggishness, 362 ; in Insanity, 568 ; Convergence Reaction with Reflex Immobility of, 644 ; Spinal Cord in Absence of Pupillary Light Reflex, 307
Pyæmia : with Meningitis, 631

RACHISTOVAINISATION : Paralysis after, 351

Recklinghausen's Disease, 623

Reflexes : Course of Afferent Portion of Arcs, 161 ; Inhibition and Excitation, 163 ; Time taken in Transmission of Impulses, 164 ; in Scarlet Fever, 183 ; Foot-Clonus, 183 ; in Infancy, 237 ; Ankle-Clonus in Hysteria, 263 ; "Fly-catching" in Frog, 401 ; Dissociation of, in Hysteria, 426 ; Toe Reflex, 432 ; Unilateral Loss of Knee Jerk in Tabes, 487 ; Mechanism of Babinski's Sign, 502 ; Significance of Tendo Achillis Jerk, 504 ; Bulbo-cavernous Reflex in Dementia Præcox, 665 ; Excitation of Vaso-Dilator Nerve Fibres in Depressor, 589 ; Abdominal, 622 ; Transmission of, in Spinal Cord, 164

Regeneration : in Peripheral Segment of Nerve, 283

Respiration : in Normal and Insane, 122 ; Mechanism, after Isolation from Extrinsic Nerve Impulses, 93 ; Changes in, of Central Origin, 364 ; Loss of, in Pseudo-bulbar Paralysis, 178

Retrobulbar Neuritis in Disseminated Sclerosis, 300

Reviews : Centres Nerveux Cérébro-spinaux (Van Gehuchten), 56 ; Bericht über die Leistungen auf dem Gebiete der Anatomie des Centralnervensystems (Edinger und Wallenberg), 57 ; Psychology and Treatment of Neurotic Symptoms (Muthmann), 58 ; Freud'sche Ideogenitätsmoment, etc. (Cross), 59 ; Die Melancholie, etc. (Dreyfus), 61 ; Erkennung und Behandlung der Melan-

cholie (Ziehen), 64 ; Arbeiten aus dem neurologischen Institute (Marburg), 133 ; Arbeiten aus der deutschen psychiatrischen Universitäts-Klinik in Prag (Pick), 133 ; Progressive Allgemeine Paralyse (Krafft-Ebing), 134 ; Borderland of Epilepsy (Gowers), 134 ; Notwendige Reformen der Unfallversicherungsgesetze (Hoche), 135 ; Mouvement Mystique Contemporain (de Fursac), 135 ; Aerztliches über Sprechen und Denken (Anton), 136 ; Blood Changes in Mental Diseases (Galdi), 205 ; Outlines of Psychiatry (White), 272 ; Étude Médico-psychologique sur Thomas de Quincey (Guerrier), 273 ; Elements of Psychology (Mellone and Drummond), 319 ; Tumours of Nervous System (Bruns), 321 ; Acute Poliomyelitis (Wickman), 324 ; Heine-Medin Disease (Wickman), 324 ; Norwegian Epidemics of Poliomyelitis (Harbitz and Scheel), 326 ; Functions of Nerve Centres (Bechterew), 327 ; Treatment of Exophthalmic Goitre (Sainton and Delherme), 327 ; A Mind that Found Itself (Beers), 328 ; Medico-Psychological Study of Alfred de Musset (Odinot), 329 ; Crowd Suggestion (Gudden), 330 ; Psychology of the Moltke Case (Merzbach), 330 ; Major Symptoms of Hysteria (Janet), 374 ; Bismarck im Lichte der Naturwissenschaft (Lomer), 376 ; Titres et Travaux Scientifiques du Docteur Pierre Marie, 377 ; University of Pennsylvania, Contributions from Department of Neuropathology, 377 ; Archiv für Geschichte der Medizin (Sudhoff), 377 ; Classification of Criminals (Ingegnieros), 444 ; Influence of Alcohol, etc., on Fatigue (Rivers), 445 ; Nuclei of Spinal Cord (Jacobsohn), 446 ; Structure of Nervous Central Organs (Edinger), 516 ; Anatomy of Brain and Cord (Harris Santee), 518 ; Modern Clinical Medicine—Diseases of Nervous System, 519 ; Psychology and Psychiatry in Hesse (Balser, Aull, and Waldschmidt), 519 ; Simulation of Insanity (Mairet), 671 ; Diseases of Spinal Cord (Williamson), 674 ; Diseases of Nervous System (Campbell-Thomson), 675 ; Manual of Psychiatry (Roques de Fursac), 731

Rheumatism : Neuritis in Scarlatinal, 649

SCARLET Fever : Reflexes in, 183 ; Hemiplegia following, 530 ; Neuritis in

- Scarlatinal Rheumatism, 649; and Uræmia, 724
- Sciatica, 430; and Disease of Hip-Joint, 313
- Sclerosis: Amyotrophic Lateral, 99; Tuberosa, 292; Cerebral, of Pseudo-Bulbar Type in Children, 357
- Scoliosis: in Infantile Paralysis, 286
- Sea-Sickness: Pathogeny and Treatment of, 311
- Senescence: Relation of Family Diseases to Premature Physiological, 595
- Sensation: Conduction of in Spinal Cord, 703; Disturbances of Cerebral Origin and Spinal Type, 499; Integrity of, in Lesion of Left Parietal Lobe, 501; Bone, 501; Phrictopathic, 562; Pain Sensation in Comparison of Spinal Cord, 414; Upright Position maintained by Sensation from Joints (?), 162
- Sensibility: Effect of Mental Work on Auditive, Visual and Tactile, 347
- Sera: "Neurotoxic" and Lesions induced by them, 27, 345; Serodiagnosis in Psychiatry and Neurology, 651; Serum Diagnosis of Syphilis, 298; Wassermann's Serum in Diagnosis of, 261, 262; Serum Treatment of Diphtheritic Paralysis, 728; Effect of Cholesterin on, 480
- Shock: Vasomotor Neurones in, 163
- Sinuses: Acute Suppuration of Sphenoidal, 423; Amblyopia after Suppuration of Accessory, 437; Operation in Chronic Inflammations of Frontal, 559; Sinus Thrombosis after Purulent Otitis, 181; Thrombosis of Superior Longitudinal and Lateral, 719; Thrombosis of Cavernous, 720
- Skull: Injuries of, 100; Tower-shaped, 550
- Sleep, 21; Experimental Study of, 408, 477, 592; Recurrent Autohypnotic, 562; Morbid Somnolence, 311
- Somnambulism: Spontaneous, 263
- Spasmodic Contraction of Finger: cured by stretching Median Nerve, 271
- Speech: Disturbances of, in Functional Psychoses, 428: *vide* Aphasia
- Spina Bifida: Treatment by Drainage of Cerebral Subdural Space, 667
- Spinal Cord: *Anatomy*: Nuclei of Human (Review), 446; Resemblances between Human and Animal, 470; Mechanism and Function, etc. (Review), 518; Middle Cells of Grey Matter, 584
- Spinal Cord: *Physiology*: Affinity for Strychnine and Cocaine, 94; Time taken in Transmission of Reflex Impulses in, 164; Reflex Excitability after Cerebral Anæmia, 344; Sensory Conduction in, 703
- Spinal Cord: *Pathology*: Changes in Muscular Dystrophy, 137; in General Paralysis, 168; Peripheral Degeneration Revealed by Longitudinal Section and Axis-Cylinder Stain, 173; in Absence of Pupillary Light Reflex, 307; after Nerve-Crossing and Nerve-Grafting, 405; Lesions in Experimental Caisson Disease, 479; Posterior Column Degenerations following Injury to Posterior Roots of Seventh Cervical Nerves, 643; Histology of Lymphogenous and Hæmatogenous Toxic Lesions of, 593; Lesions in Anterior Horn Cells in Nervous Arthropathies, 594
- Spinal Cord: *Clinical*: Subacute Combined Degeneration, 413; Pain Sensation in Compression of, 414; Diseases of the (Review), 674; Disease of Primary Motor Neurones from Poisoning by Cyanide of Potassium, 555; Pseudo-systemic Disease of, 608; Unilateral Cauda Equina Syndrome, 611; Idiopathic "Meningitis Spinalis Serosa Circumscripta," 710; Traumatic Affections of, 711
- Spine: Ankylosing Diseases of, 12, 65; Sprain of, 556
- Spondylitis: after Dengue, 115; Gonorrhœal, 193
- Stereognosis and Symboly in Lower Extremities, 502
- Stovaine Anæsthesia: Changes in Nervous System after, 705
- Strychnine and Reflex Inhibition of Skeletal Muscle, 24; Affinity of Spinal Cord for, 94; Contraction of Muscle in Poisoning by, 473; Action of, 640; Antagonistic Action of Parts of Brain to, 641
- Stupor: in Alcoholism, 258
- Subacute Combined Degeneration of Spinal Cord, 413
- Syphilis: Cerebro-spinal or Disseminated Sclerosis, 34; General Paralysis and Cerebral, 46; Epiconus Symptom-Complex in Cerebro-Spinal, 77; Neurofibrils in Cerebral, 97; Syphilogenous Diseases of Central Nervous System, 104; Mercury Treatment and Metasyphilis of Nervous System, 127; Wassermann's Serum, Diagnosis of (2), 261, 262, 572; General Paralysis in Third Year of, 267; Ascending Paralysis in, 275; Multiple Lesions, 293; Serum Diagnosis of, 298; Congenital, 300; Precocious Hemiplegia in Second-

- ary, 659; Nervous Syndrome in Secondary, 662; Syphilitic Meningitis, 174, 419; Syphilitic Spinal Paralysis, 488; Cerebro-spinal Fluid in Congenital, 297
- Syphilomania and Syphilophobia, 442
- Syringobulbia, 172, 555, 652
- Syringomyelia: Lesion in Catatonic Stupor, 26; and Acromegaly, 108; and Cheiromegaly, 172; with Bulbar Phenomena and Trophic Disturbances, 172; Case of Spasmodic (?), 390; with Syringobulbia, 555; Cervico-Bulbar, commencing with Hiccough, 652; Segmental Hypertrophy of Arm in, 653
- TABES Dorsalis:** Postcentral Cortex in, 5; with Diseases of Heart and Vessels, 35; Pruritus in, 100; with Periodic Melancholia, 124; Peripheral Neuritis resembling, 244; Some Years after Infection, 246; Osteo-Arthritic Manifestations of, 286; Syphilitic, 293; Re-educative Treatment of, 351; Gait in, 409; Juvenile, 412, 413; Unilateral Loss of Knee-Jerk in, 487; Pathogenesis of, 551; without Lightning Pains, 553; in Mother and Syphilis in Infant, 553; Etiological Treatment of, 554; Laryngeal Crisis and Paresis of Abductors of Vocal Cord in, 652; Cerebro-spinal Fluid in Diphtheritic Pseudo-Tabes, 712; Ocular Manifestations of, 605; associated with Trophic Changes suggesting Acromegaly, 607; Pruritus in, 607
- Tachycardia: Paroxysmal, 45
- Temperature and Excitability, 165
- Tendon Operations: in Spinal and Cerebral Palsies, 203, 317
- Tetany: Tetanoid States in Childhood, 44; Hysterical Pseudo-, 192
- Thrombosis: Sinus, after Chronic Purulent Otitis, 181; of Superior Longitudinal and Lateral Sinuses, 719; of Cavernous Sinuses, 720
- Thyroid: Vaso-Motor Innervation of, 546; Hypertrophy of Pituitary after Excision of, 546; in Insanity, 664; Parathyreogenic Laryngospasm, 433
- Thyroidism: Blood in, 721
- Tic-Doloureux: *vide* Neuralgia
- Tinnitus: and Vertigo, etc., 646; Division of Auditory Nerve for, 730
- Tongue: Synchronous Movements of Lower Jaw and, 35
- Tonillitis: Acute Poliomyelitis following, 709
- Torticollis: 362; Mental, 372, 618; Surgical Treatment in Mental, 372, 725
- Trauma: Skull Injuries, 100; in Etiology of Nervous Diseases, 117; Acute Psychoses in, 201; Cranial and Mental Disorder, 369; Traumatic Affections of Spinal Cord, 711
- Treatment: of Eclampsia, 53; of Arteriosclerotic Atrophy of Cerebrum, 54; of Neuralgia, 54, 55; of Neurotic Symptoms (Review), 58
- Tremor: Post-Apoplectic, 180; Hereditary, 623
- Trophœdema: Chronic, 364
- Tuberculin: Ophthalmo-Reaction to, 117
- Tuberose Sclerosis, 292
- Tumours: *Brain:* of Pituitary Duct, 110; Cortical Changes in, 169; with Jacksonian Spasm, etc., 178; Tubercle, in Childhood, 181; of Nervous System (Review), 321; Hæmangioma in Pons Varolii, 352; Diagnosis of, 422; in Cerebello-Pontine Angle, 494; Symptoms of Pontine, 495; Cerebellar, with Proptosis, 496; Papilloma of Choroid Plexus, 496; of Pons, complicated Middle Ear Suppuration, 558; of Frontal Lobe Simulating Paresis, 560; Case of Intracranial, 577; of Central Nervous System, 612, 677; Localised and Removed, 614; Cerebellar and Extra-Cerebellar, 718
- Tumours: *Spinal:* Surgical Treatment of, 128; of Cauda Equina and Lower Vertebrae, 287; of Central Nervous System, 612, 677; Surgical Treatment of, 729
- Türk: Origin of Bundle of, 91
- Typhoid: Epileptiform Convulsions and Hemiplegia in, 37, 177; Bacilli in Cerebro-spinal Fluid in, 491, 597; Lumbar Puncture in Meningeal Forms of, 557; Intracranial Abscess from Typhoid Bacillus, 249; Meningitis, 597
- ULNAR Nerve:** Occupation Neuritis of, 709
- Uræmia: Aphasia and Hemiplegia in, 560; Primary Lesions of Nerve Fibres in, 707; Postscarlatinal Convulsive, 724
- VAGUS Nerve:** Injuries of, 434, 645
- Vertigo: and Tinnitus, etc., 646; Division of Auditory Nerve for, 730
- Vestibular Apparatus: Reflex Nystag-

mus in „Diagnosis of Condition of, 436	Wedensky Inhibition, 24
Vocal Cords : Paresis of Abductors of, in Tabes, 652	X-RAYS : Action on Eye in Course of Development, 242
WASSERMANN'S Serum Diagnosis of Syphilia, 261 (2), 262, 572	ZONA : <i>vide</i> Herpes

INDEX OF AUTHORS.

- ABADIE** and **Nogue**. Tabes without Lightning Pains, 553
Abraham, K. Sexual Traumata as Form of Infantile Sexual Activity, 50
Achard. Gluteal Herpes after Lumbar Puncture, 38
Ackermann. Skull Injuries, 100
Acuna. Acute Polio-Encephalomyelitis in Boy of Three, 492
Adam, James. Spasmodic Contraction of Finger cured by Stretching Median Nerve, 271
Adamkiewicz. Pseudo-Hysterical Hemiplegia, 253
Alder, S., and Kurt Mendel. Serous Spinal Meningitis, 650
Alcock and Lynch. Physical, Chemical, and Electrical Properties of Nerves, 25
Alessi. Lesions of Cortex in Alcoholics, 346
Alexander, G. Surgery of Otogenic Meningitis, 668
Alexander and Obsteiner. Cochlear Nerve in Internal Auditory Meatus, 236
Alger. Migraine and Treatment of Eyes, 565
Alquier and Raymond. Pseudo-Bulbar Paralysis, 179; Rleklinghausen's Disease, 623
Allen. Diagnostic Sign in Recurrent Laryngeal Paralysis, 365
Amberg, Emil. Cerebral Abscess, 720
Anglade and Calmettes. Senile Cerebellum, 346
Anglade and Jacquin. Dementia Præcox, with Autopsy, 665
Anton. Sprechen und Denken (Review), 136; Psychic Disturbances in Unilateral Nasal Obstruction, 627
Apert, M. E. Amaurotic Family Idiocy, 186; Herpes Zoster and Mumps, 560
Archambault, La Salle. Acute Anterior Poliomyelitis in Adult, 605
Artom and Lhermitte. Syringomyelia, with Cheiromegaly, 172
Atlee and Mills. Brain Tumour, with Jacksonian Spasm, etc., 178
Atwood. Influence of Occupation in Nervous Disorders, 201
Audry. Syphilomania and Syphilophobia, 442
Aull, Balser, and Waldschmidt. Alcoholism, etc. (Review), 519
Austregesilo and Gotuzzo. Mental Disorders of Anchylostomiasis, 268
Ayer, J. B., and Henry Cotton. Cytological Study of Cerebro-Spinal Fluid, 207
BABINSKI. Alcoholic Neuritis, 98
Bachmann. General Paralysis in Children, 266
Bade. Tendon Operations in Spinal and Cerebral Palsies, 203
Ballet and Barbé. Syphilitic Meningitis, 419
Baldi. Abdominal Reflex, 622
Ballance, Charles A. Division of Auditory Nerve for Painful Tinnitus, 730
Ballet and Laignel-Lavastine. Old Myopathy, 349
Balser, Aull, and Waldschmidt. Alcoholism, etc. (Review), 519
Barbé and Ballet. Syphilitic Meningitis, 419
Barbé and Deny. Syringomyelic Lesion in Catatonic Stupor, 26
Barié and Lian. Epileptiform Convulsions and Hemiplegia in Typhoid, 37
Barile. Optic and Oculomotor Neuritis following Gonorrhœa, 485
Barrett and Mitchell. Posterior Column Degenerations following Injuries to Posterior Roots of Seventh Cervical Nerves, 643
Barschinger. Sciatica, 430
Barth. Organic and Functional Aphonia, 113
Bartlett and Holt. Epidemiology of Acute Poliomyelitis, 602
Basler, Adolf. Contraction of Frog's Muscle in Strychnine Poisoning, 473
Bayliss, W. M. Vaso-Constrictor Fibres in Chorda Tympani Nerve, 589; Excitation of Vaso-Dilator Nerve Fibres in Depressor Reflexes, 589
Beadles. Aneurisms of Larger Cerebral Arteries, 36
Bechterew. Functions of Nerve Centres (Review), 327
Beduschi. Acromegaly with Osteoarthropathies and Paraplegia, 256

- Beck, A. Fatiguability of Nerves, 472
 Beers. A Mind that found Itself (Review), 328
 Beevor, C. E. Pseudo-Bulbar Paralysis with Loss of Voluntary Respiration, 178; Co-ordination of Single Muscular Movements in Central Nervous System, 702
 Bellay and Tribondeau. Action of X-Rays on Eye in Course of Development, 242
 Benigni and Zilocchi. Dementia Præcox, 569
 Bennecke. Epidemic Cerebro-Spinal Meningitis, 39
 Berger, Hans. Climacteric Psychoses, 123
 Berkeley. Parathyroid Gland in Treatment of Paralysis Agitans, 131
 Bernhard, Nobécourt and Harvier. Post-Scarlatinal Convulsive Uræmia, 724
 Bernhardt. Facial Paralysis, 362
 Bernstein. Value of Lumbar Puncture, 175
 Bethe. Conducting Function of Neurofibrils, 242
 Biach, Paul. Resemblances between Human and Animal Spinal Cords, 470
 Bianchi. Development of Nerve Centres in Vertebrates, 698
 Biehl. Relation between Labyrinth and Eye, 92
 Bienfait. Torticollis, 362
 Bikeles. Spinal Cord after Nerve-Crossing and Nerve-Grafting, 405
 Bikeles and Fromowicz. Afferent Portion of Reflex Arcs, 161
 Bioglio. Urinary Constituents in Hemispheres, 35
 Blanchetière, Claude, and Schmieregeld. Serum of Epileptics, 365
 Bleuler and Jung. Dementia Præcox, 367
 Bloch, Ernst. Freud's Sexual Theory of Neuroses, 192
 Bloch and Hechinger. Anosmia in Temporo-Sphenoidal Abscess, 654
 Bokay, von. Lumbar Puncture in Cerebro-Spinal Meningitis, 53
 Bolton, Charles, and S. H. Brown. Pathological Changes in Experimental Diphtheria, 29
 Bono, A. M. Family Spastic Paraplegia, 352; Trophic Disorders in Hysteria, 426
 Booth, J. Arthur. Myasthenia Gravis Pseudo-Paralytica, 708
 Boschi. Nervous Syndrome in Secondary Syphilis, 662
 Boston, L. Napoleon. Delirium Tremens, 200
 Bouchaud. Amyotrophic Lateral Sclerosis, 99
 Bouchut and Mouriquand. Heart Failure in Exophthalmic Goitre, 566
 Boulenger. Chronic Alcoholism in a Child, 366
 Bourilhet. Paralysis Agitans in Insane Epileptic, 315
 Boycott and Damant. Spinal Lesions in Caisson Disease, 479
 Bradford, John Rose. Certain Aneurisms of Cerebral Vessels, 720
 Bramwell, Byrom. Analysis of 76 cases of Poliomyelitis Anterior Acuta, 554
 Bramwell, Edwin. Intracranial Tumour, 577
 Bratz. Etiology of Epilepsy, 721
 Bregman. Acute Ataxia, 111; Total Anæsthesia, 500
 Brissaud and Sicard. Trigeminal Neuralgia treated by Injections of Alcohol, 202
 Brown, R. Dods. Psychoses of Influenza, 439
 Brown, S. H., and Charles Bolton. Pathological Changes in Experimental Diphtheria, 29
 Browne, J. G. Epidemic Cerebro-spinal Meningitis, 353
 Bruce, Alexander. Spasmodic Syringomyelia (!), 390; Aneurism of Internal Carotid Artery and Cavernous Sinus, 462
 Bruce, Alexander, and J. H. Harvey Pirie. Origin of Facial Nerve, 685
 Bruce, Pirie, and Macdonald. Aneurism of Anterior Cerebral Artery, 449
 Bruce, W. Ironside. Sciatica and Hip-joint Disease, 313
 Bruns. Surgical Treatment of Spinal Tumours, 128; Tumours of the Nervous System (Review), 321; Idiopathic "Meningitis Spinalis Serosa Circumscripta," 710
 Buchanan, Florence. Time taken in Transmission of Reflex Impulses, 164
 Bumke. Diagnostic Meaning of Pupillary Symptoms, 265; Pupillary Movements, 301
 Bumm. Treatment of Eclampsia, 53
 Burnand. Unilateral Loss of Knee Jerk in Tabes, 487
 Burr, C. W. Mental State in Chorea, 568
 Byszowski. Cutaneous and Tendon Reflexes in Infancy, 237
 CAJAL, S. R. Reticular Apparatus of Golgi-Holmgren, 90
 Calligaris. Cells of Locus Cæruleus and Substantia Nigra, 701

- Calmettes and Anglade. Senile Cerebellum, 346
- Camp, Carl D. Traumatic Cervical Myelomalacia, 711
- Cantley. Cerebro-spinal Meningitis, 103
- Carles and Rocaz. Lumbar Puncture in Meningeal Forms of Typhoid, 557
- Carmichael, E. Scott. Leptothrix Infections, 631
- Caro. Blood in Exophthalmic Goitre and Thyroidism, 721
- Carpenter, George. Chorea with Double Optic Neuritis and Hyperpyrexia, 44
- Carr, Harvey. Psycholeptic Attacks of Hysterical Origin, 264
- Carraro and Salviolo. Physiology of Pituitary Body, 641
- Carver and Fairbairn. Hæmorrhage into Pons Varolii in Eclampsia, 250
- Cassirer and Loeser. Rotatory Movements and Nystagmus, 306
- Cassirer and Maas. Chronic Anterior Poliomyelitis, 709
- Catola. Amyotrophic Polyneuritis, 487
- Cazacon and Parhon. Chronic Trophœdema, 364
- Celler and Mandelbaum. Pathology of Myasthenia Gravis, 408
- Cerletti. Perivascular Corpuscles in Cerebral Substance, 20
- Chabbert. Acute Aseptic Meningitis, 714
- Champy and Etienne. Cellular Lesions of Anterior Horns in Nervous Arthropathies, 594
- Chardinal and Guimaraes. Pupillary Phenomena in Insane, 568
- Charpentier and Dupouy. Cranial Traumatism and Mental Disorder, 369
- Charpentier and Dupré. Chronic Psychopolyneuritis, 371
- Chartier and Sollier. Cervico-Bulbar Syringomyelia commencing with Hiccough, 652
- Chavernac. Double Optic Neuritis following Varicella, 618
- Chéné. Diphtheritic Paralysis, 170
- Cimorini. Hypertrophy of Pituitary after Excision of Thyroid, 546
- Claisse and Toltrain. Acute Syphilitic Meningitis, 290
- Claparède. Definition of Hysteria, 41 ; Psychological Methods, 549
- Clark, L. Pierce. Nocturnal Petit Mal Cured by Large Doses of Bromide, 202
- Clark, L. Pierce, and Tyson. Eye Syndrome of Dementia Præcox, 570
- Claret and Landowski. Polynucleosis of Cerebro-Spinal Fluid in Tubercular Meningitis, 39
- Claude and Raymond. Pontine Tumours, 495
- Claude and Levi-Valeusi. Juvenile General Paralysis, with Apraxic Symptoms, 666
- Claude, Schmiergeld, and Blanchetière. Serum of Epileptics, 365
- Clérambault, G. G. de. Psychopathic Intoxication with Transformation of Personality, 47
- Clot, R. Tuberculous Meningitis in Infants, 291
- Cocks and MacKenty. Headaches from Pathological Condition of Nose, etc., 386
- Collier, James, and S. A. K. Wilson. Amyotonia Congenita, 481
- Collins, Joseph. Acute Anterior Poliomyelitis, 30 ; Psychasthenia, 310
- Collins, Joseph, and H. S. Martland. Disease of Primary Motor Neurones, etc., 555
- Collins and Southard. Gliotic Cyst of Right Superior Parietal Lobule, 182
- Comby, J. Ophthalmo-Reaction to Tuberculin, 117
- Commandeur. Cerebral and Cerebro-Spinal Meningitis during Puerperium, 599
- Conzen. Tendo Achilles Jerk, 504
- Cornell, W. B. Cerebro-Spinal Fluid in Paresis, 121
- Cottentot and Dufour. Tabes in Mother and Syphilis in Infant, 553
- Cotton, Henry. Alcohol in Etiology of Mental Disease, 511
- Cotton, Henry A., and J. B. Ayer. Cytological Study of Cerebro-Spinal Fluid, 207
- Coughlin. Cerebral Abscess with Masked Symptoms, 424
- Courtney. Psychasthenia, 269
- Coux, R. de. Acute Syphilitic Meningitis, 599
- Cramer. Treatment of Arterio-sclerotic Atrophy of Cerebrum, 54
- Cramer and Wilson. Protagon: its Chemical Composition, etc., 164
- Crawford, Wm. Headache and Eye-strain, 428
- Cristiani. Use of Tobacco in Lucca Asylum, 629
- Crouzon and Doury. Gonorrhœal Spondylitis, 193
- Crouzon and Villaret. Acute Ascending Paralysis of Syphilitic Origin, 275
- Currie and MacGregor. Serum Treatment of Cerebro-spinal Fever in Glasgow Fever Hospital, 716

- D'ABUNDO. Word-Blindness with Agraphia in Left-handed Hemiplegia, 509
- Damant and Boycott. Spinal Lesions in Caisson Disease, 479
- Dana, C. L. Functions of Corpora Striata, 254
- Davids, H. Eye Findings in General Paralytics, 441
- Davies, H. Morriston, and G. Hall. Neuropathic Keratitis, 185
- Davis. Meningococcus Infections, 100
- Dawson, W. R. Alcohol and Mental Disease, 626
- Deane. Thrombosis of Superior Longitudinal and Lateral Sinuses, complicated by Pregnancy, 719
- Debove. Multiple Syphilitic Lesions, 293
- Debray. Conjugate Deviation of Eyes and Head, 306
- Déjerine-Klumpke, Madame. Complete Radicular Paralysis of Brachial Plexus with Oculo-Pupillary Phenomena, 624
- Delachanal and Massia. Ocular Manifestations of Tabes, 605
- Delherme and Sainton. Treatment of Exophthalmic Goitre (Review), 327
- Delille, Armand, and Giry. Cerebral Sclerosis of Pseudo-Bulbar Type in Children, 357
- Dench. Otitic Brain Abscess, 40
- Deny and Barbé. Syringomyelic Lesion in Catatonic Stupor, 26
- Deny and Maillard. Bilateral Motor Apraxia, etc., 657
- Dercum. Aphasia with Integrity of Left Third Frontal Convolution, 36; Tumour of Frontal Lobes, 560; Tabes with Trophic Changes suggesting Acromegaly, 607
- Descomps and Sicard. Mental Torticollis of Brissaud, 372
- Desplats. Segmental Hypertrophy of Arm in Springomyelia, 653
- Desplats, René. Electrical Treatment of Facial Tic-Douloureux by Introduction of Salicylic Ion, 52
- Dickson, W. Carnegie. Polyarteritis Acuta Nodosa and Periarthritis Nodosa, 193
- Dieulafoy. Multiple Relapsing Gangrene of Arms and Foot, 567
- Dighton. Progressive Ossifying Myositis in Boy of Eleven, 348
- Dijon. XVIII^e Congrès de Médecins Aliénistes et Neurologistes de France, 624
- Diller. Pontile Hæmorrhage, 295
- Dimitresco and Soutzo fils. Chronic Non-Moral Alcoholics, 512
- Dogiel. Anatomy of Spinal Ganglia (Review), 468
- Donley. Hypnoidization in Functional Psychoses, 629
- Dopter. Meningitis in Mumps, 249
- Dorado, Pedro. Asylum Treatment for Inebriates, 319
- Doury and Crouzon. Gonorrhœal Spondylitis, 193
- Dow, W. Epidemic Cerebro-spinal Meningitis, 289
- Dreyfus, Georges L. Die Melancholie, etc. (Review), 61
- Dromard. Hereditary Tremor, 623
- Drummond, Margaret, and S. H. Mallone. Elements of Psychology (Review), 319
- Dufour and Cottentot. Tabes in Mother and Syphilis in Infant, 553
- Dunn. Serum Treatment of Epidemic Cerebro-spinal Meningitis, 353
- Duperie. Uræmic Hemiplegia and Aphasia, 560
- Dupouy and Charpentier. Cranial Traumatism and Mental Disorder, 369
- Dupré and Charpentier. Chronic Psychopolyneuritis, 371
- Dutheil. Precocious Hemiplegia in Secondary Syphilis, 659
- EDINGER. Anatomy of Central Nervous Organs (Review), 516
- Edinger and Wallenberg. Report of Work on Anatomy of Nervous System (Review), 57
- Edmunds and Roth. Action of Barium Chloride on Fowl's Muscle, 705
- Egger. Bone Sensation, 501.
- Ehlers. General Paralysis Three Years after Syphilitic Infection, 267
- Einhorn. Herpes in Cerebro-spinal Meningitis, 38
- Elié. Alcoholic Epilepsy, 258
- Elliott, A. R. Incomplete Myxœdema, 565
- Emerson, K. Acute Poliomyelitis following Tonsillitis, 709
- Erb. Syphilogenous Diseases of Central Nervous System, 104
- Erben. Is Upright Position maintained from Joints?, 162
- Escherich. Tetanoid States in Childhood, 44
- Esprit. Diphtheritic Paralysis of Left Circumflex Nerve, 170
- Estrada. Peripheral Neuritis resembling Tabes, 244
- Etienne and Champy. Cellular Lesions of Anterior Horns in Nervous Arthropathies, 594
- Eulenburg. Neuralgias and their Treatment, 54
- Ewald. Scoliosis in Infantile Paralysis, 286

- Ewens. Dementia Præcox in India, 569
- FABRITIUS. Sensory Conduction in Spinal Cord, 703
- Fairbairn and Carver. Hæmorrhage into Pons Varolii in Eclampsia, 250
- Fayet. Dissociation of Reflexes in Hysteria, 426
- Feilchenfeld. Nervous Complications in Diphtheria, 664
- Fels. Clinical Course of General Paralysis, 196
- Findlay and Munro. Course of Cerebello-Olivary Fibres, 544
- Fischer. Patchy Atrophy of Medullary Sheaths in Cortex of General Paralytics, 96; Wassermann's Reaction in Syphilis, 261
- Flatau and Zylberblast. Surgical Treatment of Tumours of Cord, 729
- Fleischer. Retrobulbar Neuritis and Multiple Sclerosis, 300
- Fletcher. Growth in Muscular Efficiency after Fifty Years of Life, 132
- Flexner and Jobling. Epidemic Meningitis treated with Antimeningitis Serum, 600
- Forchheimer. Anorexia Nervosa in Children, 115
- Forli. Traumatic Softening of Corpus Callosum, 51
- Forsac, J. Rogues de. Mouvement Mystique Contemporain (Review), 135
- Foulerton, M'Cormack, and Pasteur. Acute Poliomyelitis with Diplococcal Infection of Spinal Sac, 245
- Fournier. General Paralysis in Third Year of Syphilis, 267
- Fraenkel and Much. Wassermann's Serum Diagnosis of Syphilis, 262
- Fragnito. Fibrils in Ganglion Cells of Vertebrates, 160
- Français and Jacques. Bulbo-Pontine Softening, 612
- Francesco. Pathology of Lenticular Nucleus, 26
- François-Franck and Hallion. Vaso-Motor Innervation of Thyroid, 546
- Frankenheimer. Adiposis Dolorosa, 431
- Frazier and Mills. Brain Tumour Localised and Removed, 614
- Freer. Laryngeal Crisis and Paresis of Abductors of Vocal Cords in Tabes, 652
- French and Hope. Persistent Hereditary Œdema of Lower Extremities, 663
- Frescoln. Complications of Alcoholism, 260
- Friedel. Formation of Neuroglia Pencils, etc., in General Paralysis, 168
- Friedländer. Hysteria and Freud's Psycho-Analytic Treatment, 193
- Fröhlich. Inhibitory Fibres in Peripheral Nerves, 588
- Fröhlich and Loewi. Action of Nitrates and Atropin on Autonomic Nervous System, 590
- Fromard. Apraxia and Dementia Præcox, 666
- Fromowicz and Bikeles. Afferent Portion of Reflex Arcs, 161
- Frugoni. Respiratory Changes of Central Origin, 364
- Fründ and Nonne. Pseudo-Systemic Disease of Spinal Cord, 608
- Fry, F. R. Motor Phenomena of Chorea, 499
- Fuchs, Alfred. Peripheral Facial Palsy, 171; Oculo-Motor Paralysis without Involvement of Internal Muscles, 184
- Fuchs, E. Oculo-Motor Paralysis without Involvement of Internal Muscles, 305
- Fuller. Neurofibrils in Dementia Paralytica, etc., 97
- Fumarola. Phenomenon of Charles Bell, 619; Electric and Operative Treatment of Peripheral Facial Paralysis, 727
- Fursac, J. Rogues de. Manual of Psychiatry (Review), 731; Mouvement mystique contemporain (Review), 135
- Fursac and Pascal. Adiposis Dolorosa in Dementia Præcox, 315
- GALDI. Blood in Mental Diseases (Review), 205
- Galewsky. Tabes some Years after Infection, 246
- Gehuchten, A. Van. Cerebro-spinal Nerve Centres (Review), 56; Ankle Clonus in Hysteria, 263; Anterior Poliomyelitis of Specific Origin, 554
- Giacchi. General Paralysis with Cerebral Syphilis, 46
- Gibney and Wallace. Epidemic Poliomyelitis, 171
- Gierlich. Neurofibrils of Motor Ganglion Cells, 343; Symptomatology of Cerebellar and Extra-cerebellar Tumours, 718
- Gioseffi. Herpes in Cerebro-spinal Meningitis, 103
- Giroux and Laubry. Acute Syphilitic Meningitis, 174
- Giry and Armand Delille. Cerebral Sclerosis of Pseudo-Bulbar Type in Children, 357
- Goldstein and Parhon. Paralysis of Abducens after Spinal Anæsthesia, 350

- Golgi. Method for Demonstration of Internal Reticular Apparatus of Nerve Cells, 700
- Gordon, A. Alcoholic Insanities, 123; Acute Bulbar Paralysis, 421; Integrity of Sensation in Lesion of Left Parietal Lobe, 501
- Gotuzzo and Austregesilo. Mental Disorders of Anchylostomiasis, 268
- Gowers, Sir William. Pseudo-myasthenia of Toxic Origin, 1; Borderland of Epilepsy (Review), 134; Mechanism of Nystagmus, 359
- Granés. Lunatic Asylums, 316
- Grasset and Rimbaud. Paraphasia, 621
- Graziani. Effect of Mental Work on Sensibility, 347
- Gregor, Adalbert. Sense of Time in Korsakoff's Psychosis, 627
- Grinker. Subacute combined Cord Degeneration, 413
- Gross, O. Das Freud'sche Ideogenitätsmoment (Review), 59
- Groves, E. W. Hey. Excision of Gasserian Ganglion in Trigeminal Neuralgia, 726
- Grünberger. Bronchiectasis and Cerebral Abscess, 356
- Gubb, Alfred S. Subcutaneous Injections of Air for Relief of Pain, 52
- Gudden. Crowd Suggestion (Review), 330
- Guerrier. Medico-psychological Study on Thomas de Quincey (Review), 273
- Guimaraes and Chardinal. Pupillary Phenomena in Insane, 568
- Gullan, A. Gordon. Exophthalmic Goitre and Antithyroid Treatment, 659
- Gunn, J. A. Myasthenic Reaction in the Frog, 150; Fly-catching Reflex in Frog, 401
- Günzburger. Pruritus in Tabes, 100
- Gurd and Nelles. Intracranial Abscess from Typhoid Bacillus, 249
- Guthrie, Pike, and Stewart. Reflex Excitability of Brain and Cord after Cerebral Anæmia, 344
- HAENEL. Tabetic Gait, 409
- Hafemann. Loss of Conductivity of Nerves at Increased Temperatures, 472
- Hajek. Operation in Chronic Inflammation of Frontal Sinus, 559
- Hall, George, and Morrison Davies. Neuropathic Keratitis, 185
- Hallion and François-Franck. Vasomotor Innervation of Thyroid, 546
- Halphen and Lombard. Reflex Nystagmus in Diagnosis of Functional Condition of Vestibular Apparatus, 436
- Hamilton, Chronic Progressive Chorea, 498
- Handwerck. Temporary Œdema of Optic Disc in Angioneurotic Œdema, 257
- Harbitz and Scheel. Acute Poliomyelitis, etc., 99; Epidemic Poliomyelitis in Norway (Review), 326
- Harris, D. F. Coloured Thinking, 475
- Harrison, Ross G. Living Developing Nerve Fibre, 241
- Hartenberg. Auto-Suggestion in Neurasthenia, 564
- Harvier, Nobécourt, and Bernhard. Postscarlatinal Convulsive Uræmia, 724
- Hatschek. Anatomy of Nucleus Ruber Tegmenti, 159
- Hay, John. Aphasia, 654
- Hébert. Acute Gonorrhœal Inflammation of Labyrinth, 647
- Hechinger and Bloch. Anosmia in Temporo-Sphenoidal Abscess, 654
- Hecht, D'Orsay. Deep Alcohol Injections for Trifacial Neuralgia, 131; Treatment of Chorea Minor, 132; Morbid Somnolence, 311
- Hegener. Toxic and Infectious Neuritis of Auditory Nerve, 255
- Heilbronner. Hysteria and Litigious Insanity, 48
- Heiman. Technique of Lumbar Puncture, 177
- Hemenway. Acute Meningitis of Convexity, 416
- Henry and Rosenberger. Purulent Cerebro-spinal Meningitis caused by Typhoid Bacillus, 597
- Herring, P. T. Development of Mammalian Pituitary, 340; Histological Appearances of Mammalian Pituitary, 341; Physiological Action of Extracts of Pituitary, 344
- Hildebrandt. Dyspraxia with Left-sided Hemiplegia, 511, 560; Surgical Treatment of Exophthalmic Goitre, 669
- Hochhaus. Brain Pathology, 404
- Hoche. Notwendige Reformen der Unfallversicherungsgesetze (Review), 135
- Holland, G. Thurstan. X-Ray Treatment of Exophthalmic Goitre, 566
- Holmes, Gordon. Postcentral Cortex in Tabes Dorsalis, 5; Spinal Changes in Muscular Dystrophy, 137; Classification of Cerebellar Disease, 420
- Holt and Bartlett. Epidemiology of Acute Poliomyelitis, 602
- Hoover. Sign for Detection of Malingering and Functional Paresis of Lower Extremities, 662

- Hope and French. Persistent Hereditary Oedema of Lower Extremities, 663
- Hosch. Delirium Tremens after Withdrawal of Alcohol, 47
- Hosford and Parkinson. Cerebellar Tumour with Proptosis, 496
- Hoskins and Southard. Cell Findings in Soft Brains, 168
- Huber, Francis. Meningococcus Hydrocephalus, 418
- Hudovernig. Central and Peripheral Facial Paralysis, 484, 545
- Hunt, J. Ramsay. Otagia, 187; Poliomyelitis Posterior of Genuiculate Ganglion, 485; Herpetic Inflammations of Genuiculate Ganglion, etc., 648; Occupation Neuritis of Deep Palmar Branch of Ulnar Nerve, 709
- INGENIEROS, José. Mental Alienation and Crime, 126; Liberation of Criminal Lunatics, 126; Insane and Penal Law, 127; Insanity, Simulation and Criminality, 313; New Classification of Criminals (Review), 444
- Ingraham and Steiner. Epidemic Cerebro-Spinal Meningitis in Hartford, 288
- Isselin. Attention Neurosis, 723
- JACQUES and Français. Bulbo-Pontine Softening, 612
- Jacquin and Anglade. Dementia Præcox, with Autopsy, 665
- Jacoby. Psychiatric Expert Evidence, 316
- Jacobsohn. Nuclei of Human Spinal Cord (Review), 446
- Jahnel. Hysterical Mutism, 504
- Jahrmärker. Final Stages of Dementia Præcox, 570
- Janet, Pierre. Hysteria (Review), 374
- Jobling and Flexner. Epidemic Meningitis treated with Antimeningitis Serum, 600
- Jochmann and Winkler. Traumatic Affections of Cord, 711
- Joffroy. Juvenile General Paralysis, 625
- Johnston, Henry M. Cutaneous Branches of Posterior Primary Divisions of Spinal Nerves, 699
- Johnston, R. H. Obstruction in Nose or Throat as Cause of Nervous and Mental Diseases, 117
- Jones, Ernest. Mechanism of Severe Briquet Attack, 114; Articulatory Capacity for Consonantal Sounds in Children, 243, 548; Side Affected by Hysterical Hemiplegia, 293; Allochiria, 294; Juvenile Tabes, 413; Phrictopathic Sensation, 562
- Jones, H. Lewis. Cervical Ribs and Atrophy of Intrinsic Muscles of Hand, 191
- Jones, Robert. Arthrodesis in Paralysis, 317
- Jong, De Josselin de. Gonorrhœal Meningitis, 598
- Joris. Posterior Lobe of Pituitary Gland, 343
- Jung. Freud's Theory of Hysteria, 427
- Jung and Bleuler. Dementia Præcox, 367
- Jung and Ricksher. Galvanic Phenomenon and Respiration in Normal and Insane, 122
- KAPPAS. General Paralysis in Senile Period, 263
- Kappers. Structure and Function of Autonomic Nervous System, 544
- Karplus and Spitzer. Experimental Lesions at Base of Brain, 169
- Kauffmann. Cholin in Cerebro-spinal Fluid, 298
- Kennedy, Robert. Section of Posterior Primary Divisions of Upper Cervical Nerves in Spasmodic Torticollis, 725
- Ker, C. B. Treatment of Cerebro-spinal Meningitis with Flexner's Serum, 715
- Kilvington and Osborne. Axon Bifurcation in Regenerated Nerves, 402
- Kleist. Psychical Disturbances in Sydenham's Chorea, 188
- Kluge. Co-operation of Alienist in Care of Defectives, 373
- Knapp, Albert. Hysterical Pure Word Deafness, 113; Sinus Thrombosis, etc., 181; Hypotonia, 409; Diagnosis of Cerebral Tumours, 422; Speech Disturbances in Functional Psychoses, 428
- Knapp, Philip Coombs. Heredity in Nervous Diseases, 29
- Kocher, Theodor. Blood in Exophthalmic Goitre, 660
- Kohts. Diphtheritic Paralysis and its Treatment, 728
- Kollarits. Torticollis Mentalis, 618
- Köllner. Etiology of Abducens Paralysis, 305
- Koplik. Treatment of Chorea Minor, 204
- Körner. Nuclear and Trunk Lesions of Vagus Recurrent and Oculomotor Nerves, 645
- Krafft-Ebing. Progressive General Paralysis (Review), 134
- Krause. Non-Traumatic Pseudospastic Paresis with Tremor, 112
- Kretschmer. Cerebro-spinal Fluid in Lues Hereditaria Tarda, 297

- Kroner. Wassermann's Serum Diagnosis of Syphilis, 261
 Kronthal. Sleep of Another, 21
 Krusius. Eserin in Pupillary Disorders, 303
 Kuckro. Disseminated Sclerosis, or Cerebro-spinal Syphilis, 34
 Küstner. Chronic Middle Ear Suppuration with Tumour of Pons, 558
 Kuttner and Meyer. Sensory Fibres in Recurrent Laryngeal Nerve, 238
- LACASSAGNE. Insanity of Maupassant, 574
 Lachmund. Convergence Reactions with Reflex Immobility of Pupil, 644
 Ladame and Von Monakow. Pure Aphemia, 655
 Laignel-Lavastine. Epileptiform Convulsions and Hemiplegia in Typhoid, 37; Acute Encephalomyelitis, 354; Unilateral Cauda Equina Syndrome, 611
 Laignel-Lavastine and Ballet. Old Myopathy, 349
 Langfeld. Sensitiveness to Light and Size of Pupil, 239.
 Langmead. Pupillary Signs in Chorea, 191
 Landowski and Claret. Polynucleosis of Cerebro-spinal Fluid in Tubercular Meningitis, 39
 Langley. Contraction of Muscle, 166
 Lasarew. Anterior Paræsthetic Meralgia, 245
 Laubry and Giroux. Acute Syphilitic Meningitis, 174
 Lavenson. Typhoid Meningitis, 597
 Le Breton. Spinal Sprain, 556
 Legrain. Alcohol and Insanity, 268
 Lejonne and Raymond. Syringomyelia, etc., 172
 Lemaitre and Rose. Palato-Laryngeal Hemiplegia, 493
 Lenz. Sympathetic Ophthalmia, 436
 Leopold. Osseous Plaques of Pia-Arachnoid in Acromegaly, 661
 Léri, André. Ankylosing Diseases of Spinal Column, 12, 65
 Leriche and Poncet. Rousseau's Disease, 437; Calvin's Disease, 437
 Levi, Ettore. Foot-Clonus, 183
 Levi-Valeusi and Claude. Juvenile General Paralysis with Apraxic Symptoms, 666
 Lewandowsky. Dissociation of Colour Sense by Focal Brain Disease, 291; Splitting off of Colour Sense, 561
 Lewandowsky and Stadelmann. Acute Multiple Sclerosis or Disseminated Myelitis, 33
- Lhermitte and Artom. Syringomyelia with Cheiromegaly, 172
 Lian and Barié. Epileptiform Convulsions and Hemiplegia in Typhoid, 37
 Liepmann. Alleged Word-Deafness in Motor Aphasia, 505
 Lloyd, Warren. Spontaneous Somnambulism, 263
 Loesser and Cassirer. Rotatory Movements and Nystagmus, 306
 Loewi and Fröhlich. Action of Nitrites and Atropin on Autonomic Nervous System, 590
 Lomer. Bismarck im Lichte der Naturwissenschaft (Review), 376
 Lombard and Halphen. Reflex Nystagmus in Diagnosis of Functional Condition of Vestibular Apparatus, 436
 Londe, Paul. Constitutional Asthenia, 113
 Long. Crural Monoplegia, 358
 Lourié. Eye-movements in Cerebellar Irritation, 304
 Lovett. Infantile Paralysis in Massachusetts in 1907, 604
 Lucas and Mines. Temperature and Excitability, 165
 Ludlum. Peripheral Spinal Degeneration, 173
 Lugaro. Functions of Neuroglia, 587
 Lynch and Alcock. Physical, Chemical, and Electrical Properties of Nerve, 25
- MAAS and Cassirer. Chronic Anterior Poliomyelitis, 709
 Maillard and Deny. Bilateral Motor Apraxia, 657
 Maillet. Sea-sickness, 311
 Mair, W., and J. Lorraine Smith. Principles underlying Weigert's Method, 638
 Mairet, A. Simulation of Insanity (Review), 671
 Mandelbaum and Celler. Pathology of Myasthenia Gravis, 408
 Marbé. Stereognosis and Symboly in Lower Extremities, 502
 Marburg, Otto. Arbeiten a. d. Neurol. Instit. Wien. Univ. (Review), 133
 Marchand and Ramadier. Thyroid Gland in Insanity, 664
 Margulies. Regeneration of Peripheral Nerve, 283
 Marie, P. Titles of Collected Works of (Review), 377
 Marina. Rudimentary Forms of Muscular Dystrophy (Erb), 483
 Marinesco. Plasticity and Amœboidism of Cells of Sensory Ganglia, 28
 Marinesco and Minea. Hypospinal Microsympathetic Ganglia, 234

- Marks, Swift, and Porter. Afferent Impulses and Fatigue of Vasomotor Centre, 94
- Marr, Hamilton C. Cerebro-spinal Fluid in General Paralysis, 635
- Martino. Gonorrhoeal Neuritis of Auditory Nerve, 649
- Martland, E. S., and Joseph Collins. Disease of Primary Motor Neurones, etc., 555
- Masoin. Medical Study of Chateaubriand, 573
- Massia and Delachanal. Ocular Manifestations of Tabes, 605
- Mayr. Secretion of Gastric Juice and Psychopathological Conditions, 51
- M'Callum. Colony and Bromide Treatment of Epilepsy, 563
- MacCormack, Pasteur, and Foulerton. Acute Poliomyelitis with Diplococcal Infection of Spinal Sac, 245
- Macdonald, Carlos F. Modern Care of Insane, 514
- M'Donald, Stuart. Cerebro-spinal Meningitis, 489
- Macdonald, Bruce, and Pirie. Aneurism of Anterior Cerebral Artery, 449
- M'Dougall, William. State of Brain during Hypnosia, 592
- MacGregor and Currie. Serum Treatment of Cerebro-spinal Fever in Glasgow Fever Hospital, 716
- MacKenty and Cocks. Headaches from Pathological Condition of Nose, etc., 366
- Mackenzie, Alice. Galvanic Reactions of Auditory Nerve, 308
- Macnamara, Eric D. Blood Pressure in Neurasthenia, 564
- Meens. Combined Psychoses, 513
- Meige. Mental Torticollis, 372; Revision of Hysteria, 616
- Meissner. Intermittent Exophthalmos, 435
- Mellone and Margaret Drummond. Elements of Psychology (Review), 319
- Meltzer. Optic Atrophy and "Tower-Shaped" Skull, 550
- Mendel, Kurt. Trauma in Etiology of Nervous Diseases, 117
- Mendel, Kurt, and S. Adler. Serous Spinal Meningitis, 650
- Merzbach. Psychology of the Moltke Case (Review), 330
- Meyer. Relative Eupraxia in Right Hemiplegia, 622
- Meyer, A. Traumatic Lesion of Pons and Tegmentum, 26
- Meyer and Kuttner. Sensory Fibres in Recurrent Laryngeal Nerve, 238
- Milhit and Tanon. Meningitis following Gonorrhoea, 174
- Milian. Treatment of Tabes, 554
- Mills and Atlee. Brain Tumour with Jacksonian Spasm, etc., 178
- Mills and Frazier. Brain Tumour Localised and Removed, 614
- Minea and Marinesco. Hypospinal Micro-Sympathetic Ganglia, 234
- Minea and Parhon. Origin of Superior Facial, 162
- Mineff, Michael. Floor of Fourth Ventricle, 235
- Mines and Lucas. Temperature and Excitability, 165
- Mingazzini. Paralysis after Rachistovainisation, 351; Conduction Aphasia, 507
- Mirman. Alcoholic Beverages in Etiology of Insanity, 48
- Mirte. Psychical Nature of Blepharospasm, 722
- Mitchell, John K. Landry's Paralysis, 246; Neurasthenia, 310
- Mitchell and Barrett. Posterior Column Degenerations following Injury to Posterior Roots of 7th Cervical Nerves, 643
- Miyake. Adolescent Insanity, 440
- Monakow, von, and Ladame. Pure Aphemia, 655
- Moncany, Charles. Kernig's Sign, 40
- Monro and Findlay. Course of Cerebello-Olivary Fibres, 544
- Monserat and Warrington. Paraplegia due to Intra-Medullary Lesion, 173
- Montesano. Perivascular Infiltration with Plasma Cells in Central Nervous System of Alcoholised Rabbits, 28
- Montet, C. de. Tuberosc Sclerosis, 292
- Mouriquand and Bouchut. Heart Failure in Exophthalmic Goitre, 566
- Much and Fraenkel. Wassermann's Serum Diagnosis of Syphilis, 262
- Müller, Ed. Acute Paraplegia after Anti-Rabic Inoculation, 415
- Muratow. Forced Movements in Central Lesions, 549
- Muthmann, Arthur. Psychology and Treatment of Neurotic Symptoms (Review), 58
- Näcke. Homosexuality, 372, 373
- Nageotte. Rapid Method for Staining Myeline in Nerve Fibres of Brain and Cord, 682
- Nambu. Hæmangioma in Pons Varolii, 352
- Nelles and Gurd. Intracranial Abscess from Typhoid Bacillus, 249
- Nieter. Typhoid Bacilli in Cerebro-spinal Fluid, 491

- Nobécourt, Harvier, and Bernhard. Post-scarlatinal Convulsive Uræmia, 724
- Nogue and Abadie. Tabes without Lightning Pains, 553
- Noica. Mechanism of Babinski's Sign, 502
- Nonne and Fründ. Pseudo-systemic Disease of Spinal Cord, 608
- Nutt. Orthopædic Therapy in Acute Anterior Poliomyelitis, 318
- OBSTEINER and Alexander. Cochlear Nerve in Internal Auditory Meatus, 236
- Odinot. Medico-psychological Study of Alfred de Musset (Review), 329
- Oeconomakis. Heterotopia of Nucleus Arcuatus, 168
- Oettinger. Recurrent Autohypnotic Sleep, 562
- Ónodi. Cerebral Complications of Nasal Origin, 645
- Oppenheim, H. Sacral Type of Disseminated Sclerosis, 32; Tumours of Nervous System, 612
- Oppenheim, Gustav. Pathological Anatomy of Multiple Sclerosis, 706
- Orbison. Herpes of Membrana Tympani, 619
- Ormerod. Disseminated Sclerosis, 31
- Orr and Rowa. Histology of Lymphogenous and Hæmatogenous Toxic Lesions of Cord, 593
- Osborne and Kilvington. Axon Bifurcation in Regenerated Nerves, 402
- Osterhaus. Nerve Anastomosis in Infantile Paralysis, 630
- PACKARD. Feeling of Unreality in Mental Disease, 199
- Pailhas. Double Personality following Hæmorrhage, 296
- Panella. Action of Suprarenal Principle on Muscular Fatigue, 547
- Pappenheim. Periodic Melancholia, etc., 124; Cerebro-spinal Fluid, 297
- Parhon and Cazacon. Chronic Trophœdema, 364
- Parhon and Goldstein. Paralysis of Abducens after Spinal Anæsthesia, 350
- Parhon and Minea. Origin of Superior Facial, 162
- Parkinson and Hosford. Cerebellar Tumour with Proptosis, 496
- Pascal and Fursac. Adiposis Dolorosa in Dementia Præcox, 315
- Pasteur, Foulerton, and MacCormack. Acute Poliomyelitis with Diplococcal Infection of Spinal Sac, 245
- Paterson, Peter. Drainage of Cerebral Subdural Space in Spina Bifida, 667
- Paul and Walton. Arteriosclerosis, 119
- Peabody, G. L. Cerebro-spinal Meningitis of Streptococcus Origin cured by Subdural Injection of Anti-streptococcus Serum, 417
- Pelz. Paralysis Agitans, 615
- Peters. Congenital Word-Blindness, 510
- Peterson, F., and Morton Prince. Psycho-Galvanic Reactions from Co-conscious Ideas in Multiple Personality, 478
- Petrén, Karl. Acromegaly and Syringomyelia, 108
- Pfersdorff. Forced Speech in Manic-Depressive Insanity, 369
- Pick, A. Atrophy of Occipital Lobe, 106; Asymbolia and Aphasia, 107; Disorders of Orientation, 112; Arbeiten aus der Psychiatrischen Universitäts-Klinik in Prag (Review), 133
- Pighini. Nerve Cells of Electric Lobe of *Torpedo Ocellata*, 471; Neutralising Power of Cholesterin, etc., 480
- Pike, Guthrie, and Stewart. Reflex Excitability of Brain and Cord after Cerebral Anæmia, 344
- Pilcz. Heredity, 120
- Pineles. Parathyreogenic Laryngospasm, 433
- Piper. Rate of Transmission in Medulated Nerve, 704
- Pirie, J. H. H. Middle Cells of Grey Matter of Cord, 584
- Pirie, J. H. Harvey, and Alexander Bruce. Origin of Facial Nerve, 685
- Pirie, Bruce, and Macdonald. Aneurism of Anterior Cerebral Artery, 449
- Plant. Serum Diagnosis of Syphilis, 298
- Plivard. Tuberculous Meningitis in Pregnancy, 715
- Poggio. Cortical Localisation of Asymboly, 717
- Polimanti, O. Physiology of Pons and Corpora Quadrigemina, 585
- Poncet and Leriche. Rousseau's Disease, 437; Calvin's Disease, 437
- Porter and Quinby. Vaso-Motor Neurons in Shock, 163
- Porter, Marks, and Swift. Afferent Impulses and Fatigue of Vasonotor Centre, 94
- Potts and Rhein. Post-Apoplectic Tremor, 180
- Poulard. Toxic Amblyopia, 307
- Price. Hysteria in Children, 297
- Prince, Morton. Criminal Responsibility of Insane, 125; Co-conscious Ideation, 406

- Prince, Morton, and F. Peterson. Psycho-Galvanic Reactions from Co-conscious Ideas in Multiple Personality, 478
- Provotelle and Schmieregeld. Psycho-Analytic Method and "Abwehrneuro-psychosen" of Freud, 572
- Pusateri. New Origin of Peduncular Bundle of Türck, 91
- QUENSEL. Word-Deafness, 619
- Quinby and Porter. Vaso-Motor Neurons in Shock, 163
- Quirsfeld. Fatigue Measurements in 64 School Children, 20
- RAIMANN. Homicidal Melancholics, 440
- Ramadier and Marchand. Thyroid Gland in Insanity, 664
- Ranké. Idiocy Secondary to Disease of Cerebral Vessels, 371
- Rankin, Guthrie. Infantile Paralysis, 245
- Raymond, F. Relationship of Family Diseases to Premature Physiological Senescence, 595
- Raymond and Alquier. Pseudo-Bulbar Paralysis, 179; Recklinghausen's Disease, 623
- Raymond and Claude. Pontine Tumours, 495
- Raymond and Lejonne. Syringomyelia, etc., 172
- Rebaud. Pruritus in Tabes, 607
- Redlich. Cortical Changes in Cerebral Tumours, 169; Peculiar Pupillary Phenomenon, 303
- Rehm. Cytological Examination of Cerebro-spinal Fluid, 713
- Reich. Injuries of Vagus Nerve, 434
- Reichardt. Brain Matter, 697
- Reiss. Paranoid Symptom-Complexes in Degenerates, 196
- Remenar. Spinal Anæsthesia, 204
- Renner. Localisation of Pain in Compression of Cord, 414; Syphilitic Spinal Paralysis, 488
- Rennie, George E. Exophthalmic Goitre with Myasthenia Gravis, 229
- Rénon. Polyglandular Syndromes and Opothrapy, 669
- Renterghem, A. W. van. Psychotherapy, 55
- Réthi. Amblyopia after Accessory Sinus Suppuration, 437
- Retzlaff. Diagnostic Value of Pupillary Immobility and Sluggishness, 362
- Rhein, John S. W. Syringomyelia, with Syringobulbia, 555; Apraxia, with Autopsy, 658
- Rhein and Potts. Post-Apoplectic Tremor, 180
- Rhodes, J. Milson. Mentally Defective in Prison, 575
- Rickscher. Statistical Study of General Paralysis, 196
- Rickscher and Jung. Galvanic Phenomenon and Respiration in Normal and Insane, 122
- Riehm. Simulation of Insanity, 373
- Rimbaud and Grasset. Paraphasia, 621
- Rivers. Influence of Alcohol, etc., on Fatigue (Review), 445
- Robinowitsch. Diseases of Conus Medullaris, 653
- Robson. Synchronous Movements of Lower Eyelids with Tongue and Lower Jaw, 35
- Rocaz and Carles. Lumbar Puncture in Meningeal Form of Typhoid, 557
- Roënwaldt. Simplification of Nissl's Stain, 406
- Rogers and Smallwood. Studies in Nerve Cell, 233
- Rolleston. Reflexes in Scarlet Fever, 183; Hemiplegia following Scarlet Fever, 530
- Römheld. Cerebro-spinal Fluid in Diphtheritic Pseudo-Tabes, 712
- Rose. Neuritis secondary to Zona, etc., 350
- Rose and Lemaitre. Palato-Laryngeal Hemiplegia, 493
- Rosenberger and Henry. Purulent Cerebro-spinal Meningitis caused by Typhoid Bacillus, 597
- Rosenfeld. Mental Disturbances in Vaso-motor Neuroses, 296
- Rossi. "Neurotoxic Sera," 27, 345
- Rossolimo. Toe-Reflex, 432
- Roth and Edmunds. Action of Barium Chloride on Fowl's Muscle, 705
- Rothmann. Supranuclear Auditory Tracts, 402
- Rows and Orr. Histology of Lymphogenous and Hæmatogenous Toxic Lesions of Cord, 593
- Russell, Colin K. Tabes Dorsalis and its Re-educative Treatment, 351
- Russell, J. S. Risien. Diagnosis of Organic from Functional Nervous Affections, 284
- SAENGER, A. Focal Symptoms in Diffuse Brain Disorders, 359
- Sainton and Delherme. Treatment of Exophthalmic Goitre (Review), 327
- Sala. Sequel to Aseptic Lesions of Brain, 642
- Salaris, Sanna, Jacksonian Epilepsy, 425

- Salecker.** Segmental Abdominal Paralysis, 247
- Salviolo and Carraro.** Physiology of Pituitary Body, 641
- Sand, René.** New Selective Stain for Nervous System, 91
- Sano.** Action of Strychnine and Caffeine, 640; Antagonistic Action of Particular Parts of Brain to Strychnine, 641; Affinity of Spinal Cord for Strychnine and Cocaine, 94
- Santee, Harris E.** Anatomy of Brain and Spinal Cord (Review), 518
- Sarbo, Arth. v.** Landry's Paralysis, 710
- Sawyer.** Cytodiagnosis in Practical Medicine, 264
- Scarpini.** Primary Lesions of Nerve Fibres in Uræmia, 707
- Schaefer.** Dementia Præcox, Catatonic Form, 198
- Scheel and Harbitz.** Acute Poliomyelitis, etc., 99; Epidemic Poliomyelitis in Norway (Review), 326
- Schlesinger.** Spondylitis Infectiosa after Dengue, 115; Epidemic Cerebro-spinal Meningitis in Adults, 417
- Schmiergeld and Provotelle.** Psycho-Analytic Method and "Abwehrneuro-psychosen" of Freud, 572
- Schmiergeld, Blanchetière, and Claude.** Serum of Epileptics, 365
- Schmoll.** Paroxysmal Tachycardia, 45; Symptoms due to Coronary Arterio-Sclerosis, 46.
- Schneider and Vandeuve.** Serum-therapy in Diphtheritic Paralysis, 728
- Schroeder.** Pains in Manic-Depressive Insanity, 268
- Schrötter.** Exophthalmic Goitre, 363
- Schulz, Ernst.** Hysterical Hemiplegia, 357
- Schuster.** Three Chinese Brains, 697
- Schuster, Paul.** Mercurial Treatment and Metasyphilis of Nervous System, 127
- Schuyten.** Measurement of Intellectual Fatigue in Children, 407
- Schwenkenbecher.** Menthol Poisoning, 567.
- Serbsky.** Korsakow's Disease, 441
- Shaw.** Opsonic Index to Various Organisms in Control and Insane Cases, 270
- Sherren.** Neuritis of Ulnar Nerve from Deformity of Elbow Joint, 486
- Sherrington.** Strychnine and Reflex Inhibition of Skeletal Muscle, 24; Reflex Inhibition and Excitation, 163; Reciprocal Innervation of Antagonistic Muscles, 403
- Shima.** Action of Adrenalin on Nervous System, 474
- Sicard and Brissaud.** Trigeminal Neuralgia treated by Injections of Alcohol, 202
- Sicard and Descomps.** Mental Torticollis of Brissaud, 372
- Sichel.** Mental Diseases among Jews, 443
- Sidis, Boris.** Sleep, 408, 477, 592
- Siegert.** Nervousness and Nutrition in Childhood, 708
- Silberberg.** Typhoid Bacilli in Cerebro-spinal Fluid, 597
- Smallwood and Rogers.** Studies in Nerve Cells, 233
- Smith, J. Lorrain, and W. Mair.** Principles underlying Weigert's Method, 638
- Smithies.** Hemiplegia in Typhoid, 177
- Sollier and Chartier.** Cervico-Bulbar Syringomyelia commencing with Hiccough, 652
- Solomowicz.** Centre for Submaxillary Gland, 588
- Sommer.** Acute Traumatic Psychoses, 201
- Souques.** Aneurism of Sylvian Artery, 496
- Southard.** Gliosis in Acquired Epilepsy, 497
- Southard and Collins.** Gliotic Cyst of Right Superior Parietal Lobule, 182
- Southard and Hoskins.** Cell Findings in Soft Brains, 168
- Soutter, Robert.** Silk Ligaments in Infantile Paralysis, 515
- Soutzo fils.** Serum Reaction in General Paralysis by Wassermann's Method, 572
- Soutzo fils and Dimitresco.** Chronic Non-Moral Alcoholics, 512
- Spieler.** Diphtheritic Paralysis and Heart Failure in Diphtheria, 116
- Spielmeyer.** Nervous System after Stovaine Anæsthesia, 705
- Spiller, W. G.** Epiconus Symptom-Complex in Cerebro-spinal Syphilis, 77; Tumours of Cauda Equina and Lower Vertebrae, 287; Paralysis of Upward Associated Ocular Movements, 361; Occlusion of Posterior Inferior Cerebellar Artery, 557; Tumour Malformations of the Nervous System, 677
- Spitzer and Karplus.** Experimental Lesions at Base of Brain, 169
- Stadelmann and Lewandowsky.** Acute Multiple Sclerosis or Disseminated Myelitis, 33
- Starr, M. Allan.** Epidemic Infantile Paralysis, 604

- Stefani. Osteo-arthritic Manifestations of Tabes, 286
- Steiner and Ingraham. Epidemic Cerebro-spinal Meningitis in Hartford, 288
- Stelzner. Cerebellar Atrophy, 359
- Stengel. Nervous Manifestations of Arteriosclerosis, 262
- Stephenson. Lumbar Puncture in Optic Neuritis, 265; Juvenile Tabes Dorsalis, 412
- Sternberg. Muscular Strength of Hemiplegics, 253
- Stertz. Serodiagnosis in Psychiatry and Neurology, 651
- Stewart, G. N. Automatic Respiratory and Cardiac Mechanisms after Isolation from Nerve Impulses, 93
- Stewart, Purves. Disease of Post-Central Gyrus, with Astereognosis, 379
- Stewart, Pike, and Guthrie. Reflex Excitability of Brain and Cord after Cerebral Anæmia, 344
- Stillman, C. K. Post-Delirious Alcoholic Stupor, 259
- Stransky, E. Peripheral Nerves in General Paralysis, etc., 345
- Sträussler. Tumours of Pituitary Duct, 110; Disturbances of Sensation, 499
- Strümpell. Tabes Dorsalis with Diseases of Heart and Vessels, 35
- Sudhoff. Archiv für Geschichte der Medizin (Review), 377
- Swift, Porter, and Marks. Afferent Impulses and Fatigue of Vasomotor Centre, 94
- Sym, W. G. Partial Ptosis with Exaggerated Movement of Affected Eyelid, 337
- TAIT, John. Freezing of Frog's Nerve, 166
- Tamburini. Protection of Society from Criminal Lunatics, 629
- Tanon and Milhit. Meningitis following Gonorrhœa, 174
- Taylor. Graves' Diseases treated by Thyroidectomy, 362
- Taylor, F. L. Traumatic Abscess of Frontal Lobe, 492
- Tello. End-Plates of Muscle after Section of Nerves, 95
- Thomas, André. Anatomy of Peripheral Facial Paralysis and Facial Hemispasm, 167
- Thomsen. "Zwang" Phenomena, 438
- Thomson. Brain Abscess, 180
- Thomson, H. Campbell. Disease of Nervous System (Review), 675
- Thomson, St Clair. Causes and Symptoms of Thrombosis of Cavernous Sinus, 720
- Thorburn, Wm. Symptoms due to Cervical Ribs, 312
- Tillmanns. Puncture of Brain, 724
- Többen. Treatment of Epidemic Cerebro-spinal Meningitis, 104
- Toltrain and Claisse. Acute Syphilitic Meningitis, 290
- Trautmann. Acute Suppuration of Sphenoidal Sinuses, 423
- Tribondeau and Bellay. Action of X-Rays on Eye in Course of Development, 242
- Trömner. Abasia or Dysbasia, 113
- Trotter. Cerebellar Abscess, 356
- Tucker. Epilepsy—So-called Idiopathic, 498
- Turner, A. Jefferis. Infantile Paralysis simulating Meningitis, 413
- Turner, John. Structure of Grey Matter, 283
- Tyson and Pierce Clark. Eye Syndrome of Dementia Præcox, 570
- URBAN. Pseudo-Appendicitis Hysteria, 661
- VALKENBERG, Van. Projection Fibres of Occipital Lobe, 698
- Vandeuvre and Schneider. Serum-therapy in Diphtheritic Paralysis, 728
- Varendonck. Ideals of Children, 549
- Vennat. Meningitis in Mumps, 249
- Verworn, Max. Neurone Theory, 234
- Vigouroux. Papilloma of Choroid Plexus, etc., 496
- Villaret and Crouzon. Acute Ascending Paralysis of Syphilitic Origin, 275
- Vincenzo. Bulbo-Cavernous Reflex in Dementia Præcox, 665
- Vining. Acute Ascending Paralysis, 246
- Volpi-Ghirardini. Arcuate Nuclei and External Anterior Arciform Fibres of Medulla Oblongata, 471
- WALDSCHMIDT, Balser, and Aull. Alcoholism, etc. (Review), 519
- Wallace and Gibney. Epidemic Poliomyelitis, 171
- Wallenberg and Edinger. Report of Work on Anatomy of Nervous System (Review), 57
- Waller, A. D. Action of Aconitine on Nerve Fibres, 94
- Walton, G. L. Anterior Poliomyelitis in Adult, 98; Migraine, an Occupation Neurosis, 618
- Walton and Paul. Arteriosclerosis, 119
- Warda, Wolfgang. Obsessional States, 123

- Warrington, W. B. Course and Progress in Disseminate Sclerosis, 521
- Warrington and Monserrat. Paraplegia due to Intra-Medullary Lesion, 173
- Weisenburg. Lesions in Cerebello-Pontile Angle, 494
- Wendenburg. Post-traumatic, Transitory Disturbances of Consciousness, 427
- Westphal. Hysterical Pseudotetany, 192; Motor Apraxia, 357
- Weygandt. Idiocy and Dementia Præcox, 368
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- White, Wm. A. Outlines of Psychiatry (Review), 272
- Wickman, Ivar. Acute Poliomyelitis (Review), 324; Heine-Medin Disease (Review), 324
- Willerval. Tuberculous Meningitis in Infants, 598
- Williams, Tom. Pathogenesis of Tabes Dorsalis, 551
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- Wladimiroff. Scarlatinal Rheumatism, 649
- Wollstein. Cerebro-Spinal Fluid in Anterior Poliomyelitis, 651
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1908

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